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Research Article

Neuronal correlates of ADHD in adults with evidence for compensation strategies – a functional MRI study with a Go/No-Go paradigm

Neuronale Korrelate bei Erwachsenen mit Aufmerksamkeitsdefizit/Hyperaktivitäts-Syndrom (ADHS) – eine Studie mittels funktionellem MRT und einem Go/NoGo-Paradigma

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Abstract

Objective: Response inhibition impairment is one of the most characteristic symptoms of attention-deficit/hyperactivity disorder (ADHD). Thus functional magnetic resonance imaging (fMRI) during a Go/No-Go task seems to be an ideal tool for examining neuronal correlates of inhibitory control deficits in ADHD. Prior studies have shown frontostriatal abnormalities in children and adolescents. The aim of our study was to investigate whether adults with ADHD would still show abnormal brain activation in prefrontal brain regions during motor response inhibition tasks.
Methods: fMRI was used to compare brain activation in 15 untreated adult patients with ADHD and 15 healthy reference volunteers during performance of a Go/No-Go task.
Results: In contrast to various other studies with children and adolescents with ADHD, we found no significant difference in the activity of anterior cingulate cortex (ACC) or other frontostriatal structures between ADHD and healthy adults. Significantly enhanced activity was found in the parietal cortex, which is known to play an important role in building up attention.
Conclusion: We hypothesize that the enhanced activity is due to the ability of adult ADHD patients to compensate their deficits for a short time, which is demonstrated in our study by equal task performance in both groups.

Keywords: ADHD, fMRI, Go/No-Go task, neuronal compensation

Zusammenfassung

Einleitung: Die Impulskontrollstörung ist ein charakteristisches Symptom der Aufmerksamkeitsdefizitstörung (ADHS). Go/NoGo Paradigmen eignen sich hervorragend, um mittels funktioneller Kernspintomographie (fMRT) die neuronalen Korrelate der Impulskontrollstörung zu untersuchen. Frühere Studien zeigen Veränderungen in der Aktivierung frontostriataler Aktivität bei Kindern und Heranwachsenden. Das Ziel unserer Studie war zu untersuchen, ob diese Veränderungen bei Erwachsenen ebenfalls nachweisbar sind.
Methode: Mittels fMRT wurde die cerebrale Aktivität bei 15 Erwachsenen mit ADHS im Vergleich zu Kontrollprobanden mittels eines Go/NoGo-Paradigmas untersucht.
Ergebnisse: Im Gegensatz zu Untersuchungen an Kindern und Jugendlichen mit ADHS fanden wir keine signifikanten Unterschiede in der Aktivität der anterioren Gyrus cinguli (ACC) und anderen Bereichen des frontalen-striatalen Cortex. Signifikant erhöhte Aktivität fand sich im parietalen Cortex, der eine wesentliche Funktion hat bei der Aufrechterhaltung von Aufmerksamkeit.

Fazit: Die erhöhte Aktivität im parietalen Cortex könnte ein Korrelat für die Fähigkeit sein, Aufmerksamkeitsdefizite für kurze Zeit zu kompensieren.

Schlüsselwörter: ADHS, fMRT, Go/NoGo-Paradigma, neuronale Kompensation

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is among the most common behavioural disorders of childhood [1]. In about 30% of the affected children, symptoms such as inattention, impulsiveness and hyperactivity persist for the rest of their life [2]. Impulsiveness can be seen as an impairment of motor control. Therefore, tasks of motor response inhibition such as Go/No-Go, stop, and Stroop tasks are an excellent tool for examining the neuronal correlates of ADHD with functional imaging [3], [4], [5], [6]. These tasks are well established paradigms for functional magnetic resonance imaging (fMRI). In healthy volunteers they result in an activation of the right inferior frontal gyrus (IFG), right middle frontal gyrus [7], [8] and the anterior cingulate cortex (ACC),which is known to play an important role in the detection of errors [9], attentional control [10] and monitoring of response conflicts [11].

Functional imaging studies using these tasks have shown abnormalities in frontal lobe activation in children, adolescents and adults with ADHD. However, results of functional imaging studies are inconsistent [12]. Because of the large changes in brain structure and function associated with brain development and the known changes in clinical ADHD phenomenology with age [13], [14] results of functional investigations should be classified into range of age groups [15].

In children and adolescents with ADHD functional neuroimaging data indicate fronto-striatal dysfunction during inhibitory control tasks. Rubia et al. found reduced activity in the right IFG [5], [16], whereas others found enhanced activity in prefrontal areas during a response conflict task [17] or attenuated striatal activation and enhanced prefrontal activation while performing a Go/No-Go task [6]. The first functional imaging study of ADHD in adult used positron emission tomography (PET) and found reduced glucose metabolism, both global and regional [18]. The largest reductions were in the premotor cortex and the superior prefrontal cortex. Bush et al. found reduced activity in ACC during a counting Stroop task in adult ADHD patients [3]. In a working memory task with PET Schweitzer et al. found activity in the ADHD group was more diffuse and less frontal [19]. fMRI of working memory tasks showed significantly decreased activity in cerebellar and occipital regions and a trend toward decreased activation in the prefrontal cortex [20]. Beside prefrontal abnormalities, alteration of the parietal attention system comes more and more into the focus of pathophysiology of ADHD. In an event-related fMRI oddball paradigm, Tamm et al. described significantly less bilateral parietal activation in adolescent individuals with ADHD [21]. Durston et al. found decreased activity in inferior parietal cortex during a Go/No-Go task in boys with ADHD [4].

Comparisons among studies are difficult because of differences in the experimental design of the paradigm and the selection of patients. The objective of this study was to examine the neuronal correlates of adult ADHD and make it comparable with the results in children and adolescents; therefore we used the Go/No-Go paradigm which was first used by Vaidya et al. in children in which striatal activation was reduced in ADHD children [6].

Method

Subjects

15 native German speaking adults (11 male, 4 female, age range 21–42 years, mean age 28.1) recruited from our outpatient clinic for ADHD fulfilled the DSM-IV criteria for the diagnosis of ADHD and 15 age and gender matched reference subjects (11 male, 4 female, age range 21–46 years, mean age 28.8) without any history of psychiatric disease or psychiatric medical treatment participated in our study. All subjects provided written consent, and the study was approved by the Ethics Committee of the Hannover Medical School.

Inclusion criteria for all subjects were: age 18–50 years, normal or corrected to normal vision and right-handedness. For the patient group, a diagnosis of ADHD according to DSM-IV criteria was required, with childhood onset and persistence of symptoms into adulthood. Patients with a co-morbid current psychiatric diagnosis, drug abuse, medical or neurological disorder, including tics or Tourette's syndrome and head injuries, were excluded from the study.

Characteristics of the 15 subjects including demographic data and scores on the German short-version of the Wender Utah Rating Scale (WURS-k) [22], the DSM-IV [23] and Conners' Adult ADHD Rating Scales (CAARS) [24] are shown in Table 1. Controls were recruited from our department and excluded for any significant history of psychiatric illness.

Care was taken that patients with pre-study treatment stopped taking their medication at least 3 weeks before the examination, to be sure that the drug was fully washed out.

Experimental task

Stimuli were generated on a PC and projected via a Nec 2001 color projector, through a collimating lens onto a

Table 1: Characteristics and scores of the ADHD patients

Subject no.	Age (years)	Sex	WURS-k cut-off > 30 points	CAARS	DSM-IV	
1	22	m	63	positive	9	9
2	21	m	35	positive	8	3
3	40	f	48	positive	7	3
4	21	f	57	positive	7	9
5	32	m	67	positive	9	9
6	20	m	63	positive	7	8
7	23	m	32	positive	7	5
8	21	m	37	positive	8	5
9	29	m	53	positive	9	6
10	23	m	43	positive	4	7
11	37	f	71	positive	9	1
12	21	f	39	positive	7	1
13	29	m	57	positive	9	2
14	42	m	48	positive	5	8
15	41	m	44	positive	3	7

WURS-k = Wender Utah Rating Scale (indicates ADHD at a score of more than 30); CAARS = Conners Adult ADHD Rating Scale (the analysis is conducted separately with respect to sex and age and gives a hint of the subject's current state); DSM-IV = Diagnostic and Statistical Manual of Psychiatric Disorders (a score higher six in the first nine items indicates attentional problems; a score higher six in the last nine items indicates hyperactivity).

diffusing screen inside the magnet bore, so that it was comfortable to see the stimuli via a small mirror mounted above the subject's head.

Subjects performed two runs of a Go/No-Go task. After the first run, they were instructed to swap the response device from the right to the left hand. One run lasted 210 s and consisted of 10 alternating Go and No-Go blocks, each 21 s long.

For each block we used white letters as stimuli presented for 800 ms with an interstimulus interval of 200 ms duration. Depending on the block type, a different task instruction (i.e. “press for all letters” for Go blocks and “do not press for X” for No-Go blocks) were presented for 3 s before the stimulation, resulting in effective stimulus presentation time of 18 s. The letter “X” occurred within the block at a frequency of 50 percent, all other letters were presented randomly and only once per block. Subjects’ responses and their reaction times were recorded by a PC connected to the response device.

fMRI acquisition

Structural and functional MRI scans were acquired on a GE Signa 1.5-T Horizon LX System (General Electric, Milwaukee) at the Hannover Medical School, Department of Neuroradiology. A standard head coil was used for radiofrequency transmission and reception. A series of 26 axial (i.e. parallel to the bicommisural plane) spin echo T2-weighted structural images was obtained (Tr=2000ms, Te=40ms, slice thickness 5 mm, field of view 26 cm and matrix 256*256 pixels). Functional scans covering the whole brain were acquired at the same locations as the structural scans by using a multislice two dimensional echo planar imaging sequence depicting the blood-oxygenation level dependent (BOLD) signal (Tr=3000ms, Te=50ms, flip angle 90°, slice thickness 5 mm, field of view 26 cm and matrix 64*64 pixels).

Data analysis

Date were analyzed with Statistical Parametric Mapping (SPM2, Welcome Department of Imaging Neuroscience; London) employing a random-effects model. The SPM methodology is discussed in detail elsewhere [25]. The functional scans were realigned to the first volume to correct for interscan movements by means of a rigid body transformation with three rotation and three translation parameters. The functional scans were subsequently spatially normalized (Montreal Neurological Institute template) resulting in a voxel size of 2*2*2 mm and finally smoothed spatially (Gausian kernel with 6 mm full width at half maximum). A general linear model and a boxcar function were used to calculate the activation maps. The covariance of interest was estimated according to the general linear model at each and every voxel, and low frequency fluctuations were modelled as covariates of no interest. The best least square fit of the adjusted data to modelled experimental condition represent the

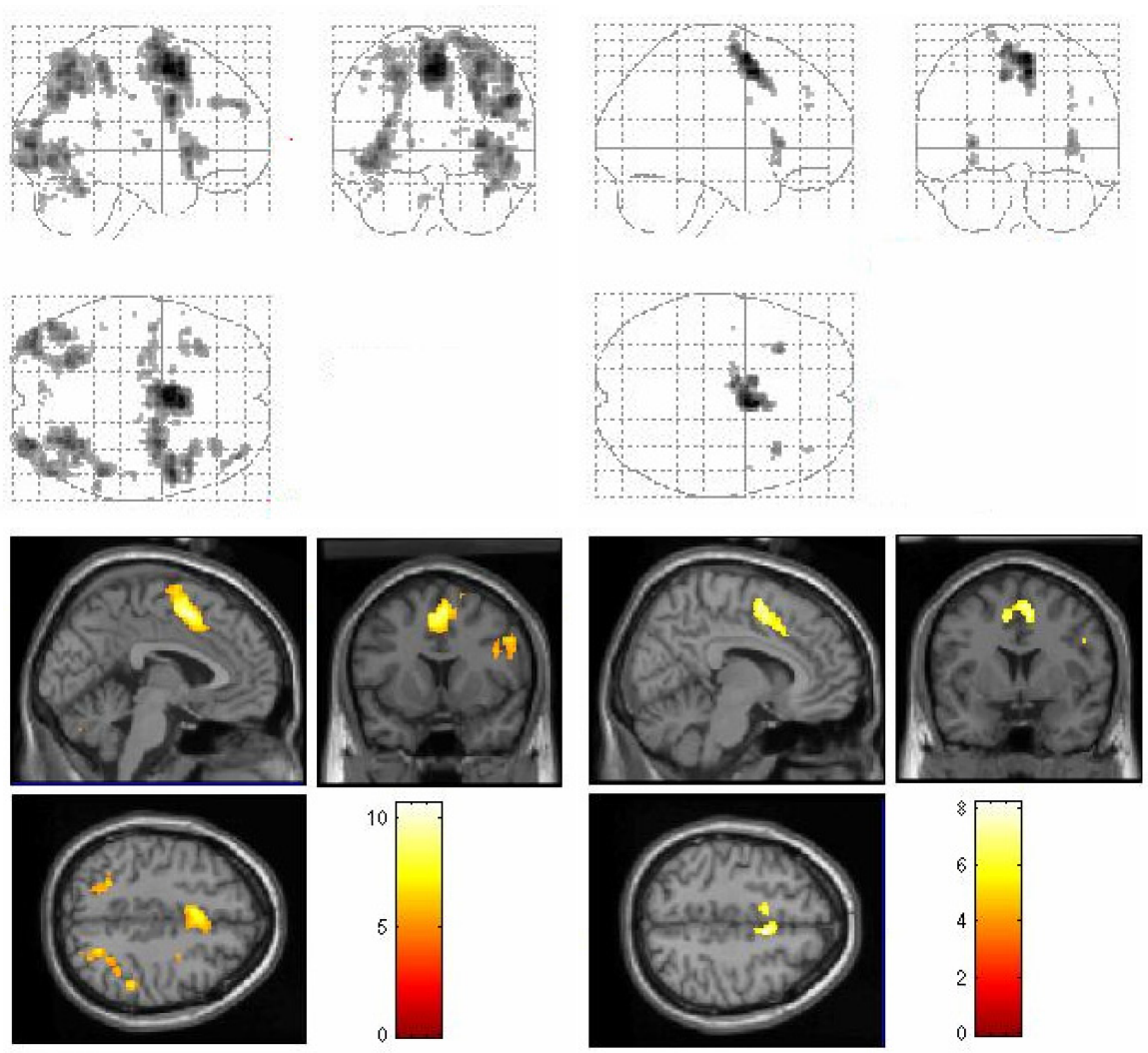

Figure 1: Group analysis of activation patterns: ADHD versus healthy subjects.
Left ADHD, right controls. Upper row: summarized activity from the whole brain. Lower row: activity is shown rendered onto a standard MRI template. Threshold for all picture is p<0.05 (corrected). Peak activity is in the ACC and additional parietal activity in the ADHD group.

parameter estimates. Significant hemodynamic changes for each contrast were assessed using Z statistical parametric maps. We report activation sites reaching a height threshold corresponding to p<0.05 corrected for multiple comparisons. Neuroanatomical locations of activation were calculated with the WFU Pick Atlas Tool (http://www.ansir.wfubmc.edu/).

First activation maps were averaged for all subjects individually and in groups in a random effect model. Finally, two-sample t-tests were performed on contrast images to investigate group differences in activation between patients and controls by a region-of-interest (ROI) analysis, implemented in the MarsBar package (http://marsbar.sourceforge.net/) to investigate changes between groups. ROIs included all voxels activated in the No-Go>Go condition at the threshold of p<0.001 [26].

Results

Behavioural data

Analysis of reaction time revealed no significant group differences in performance between adults with and without ADHD (Table 2). Both control subjects and ADHD subjects showed longer reaction times in the No-Go blocks than in the Go blocks. The number of wrong responses was 5.1±2.3 in the subject group and 4.7±2.2 in the ADHD group.

Functional MRI results

Group analysis

Both groups showed activation of the ACC (Figure 1). The peak activation was in the medial frontal gyrus and extended into ACC (Table 3). The ADHD group additionally showed activation in superior and inferior parietal lobe bilateral and in the medial and inferior occipital gyrus left.

Table 2: Performance data

	ADHD		Healthy Controls		
	Mean	SD	Mean	SD	p*
Errors					
Errors of commission press for X in No-Go blocks	4.70	2.29	5.13	2.38	0.44
Omission errors for correct letter	1.13	0.99	0.80	0.71	0.20
Reaction time					
Reaction time during No-Go blocks for correct reactions	380.53	40.11	372.17	48.01	0.34
Reaction time during Go blocks	277.73	38.71	281.33	58.90	0.76

*unpaired t-test, only corrected reactions

Table 3: Regions activated during Go/No-Go Task in ADHD and Control group

	Number of voxels activated	Maximum T-value	Peak location Talairach coordinates	Number of voxels activated	Maximum T-value	Peak location Talairach coordinates	Maximum T-value
	Regions activated in ADHD group			**Regions activated in Control group**			**Group contrasts (p<0.001)**
Superior+Inferior Parietal Lobe left	200	8.74	22 –70 40	–	–	–	4.26
Superior+Inferior Parietal Lobe right	355	8.04	30 –68 50				5.32
Anterior Cingulate Gyrus	251	6.00	–4 2 50	122	5.8	8 4 50	
Inferior+Middle+ Medial Frontal Gyrus Precentral Gyrus	1375	6.40	50 6 34	328	5.9	6 0 60	
Inferior+ Middle Occipital Gyrus left	437	7.12	30 –92 –6	–	–	–	4.23

Comparison between both groups revealed significant ($p<0.001$) differences in these areas.

Single-subject analysis

We inspected each individual case for significant brain activation ($p<0.001$) in the region of anterior cinculate cortex and superior and inferior parietal gyrus bilaterally. 14 of 15 ADHD subjects and 11 of 15 control subjects showed significant activation during No-Go condition in the region of ACC. In the parietal cortex 12 of 15 ADHD subjects and 5 of 15 control subjects showed activation.

Discussion

A Go/No-Go task was used to examine adult ADHD patients with fMRI. The neuroactivation pattern observed in adults with ADHD differed qualitatively and quantitatively from that of healthy control subjects. We chose to examine more males (11) in contrast to females (4) because according to Wender in ADHD children there is a gender ratio of male to female of about 3:1 [27]. As there are many studies with fMRI and children and hypothetically the gender ratio may persist into adulthood we wanted to have a good comparability to other studies. There are two major results in this study. Firstly in contrast to the control group we found significantly enhanced activity in parietal and occipital regions which has not been described before in Go/No-Go trials in adults with ADHD. Secondly in the patient group as well as in the control group the chosen task led to mild activation of the inferior and middle frontal gyrus and strong activation of ACC.

Between groups analysis showed that the activation did not differ significantly. The results have to be discussed in terms of

- the chosen paradigm
- difference between ADHD in adults and children respectively adolescents
- difference between adult ADHD and controls.

The block design of the paradigm we used is not very elaborated compared to others used nowadays but the main target of this study was to make it comparable with studies already done in children. The block design leads to uncertainty in the interpretation of results because activation in the experimental blocks may not only reflect response inhibition but also changes in stimulus analysis, response preparation and processing of conflicts [28]. Block designs are also known to be susceptible to habituation and changes in behavioural strategies between blocks [29]. Reaction time during Go blocks was much faster than during No-Go blocks. Although always described as Go/No-Go paradigm in the literature the high ratio of Go to No-Go trials leads to the possibility that activation related to No-Go blocks is due to selective attention rather than response inhibition [28]. Therefore it has little impact on response inhibition and much more on selective attention. This could reflect the low activity of inferior and middle frontal gyrus and the high activation of ACC found in this study. Despite this limitation due to the paradigm we have to explain the differences of results between adults and children respectively adolescents in which frontal activity differs between healthy and ADHD subjects. In an fMRI study with working memory in adult ADHD patients Valera et al. expected altered activation in the prefrontal cortex. But they only found a trend toward decreased activation which is not significant [20]. Bush et al. compared adult ADHD patients and healthy volunteers in a countingstroop experiment and they found no activity in ACC but enhanced activity in frontostriatal networks [3]. They conclude that ADHD patients might compensate by recruiting a different, less efficient pathway. Developmental factors may be the reason of different activation of brain regions in ADHD. Most fMRI studies on ADHD are performed with children and adolescents. Konrad et al. could show that there are differences in attentional networks between children and adults [30]. Children showed significant reduced activity in prefrontal and temporo-parietal regions during attentional tasks and they conclude that the differences are due to developmental changes and cognitive strategies. Fassbender et al. suggested that ADHD should be characterized not only by neuronal hypoactivity as it is commonly thought, but neuronal hyperactivity as well, in regions of the brain that may relate to compensatory brain and behavioural functioning. As a consequence of delayed development a lack of coordination of higher order regions may result in more effortful and in some cases less accurate responding and processing of stimuli [31]. In the present study, behavioural data (reaction time and number of correct responses) did not differ in the two groups. In spite of clinical symptoms like attention deficit, patients were able to concentrate for a short while. From the clinical point of view this is a well known ability. But obviously patients are not able to keep concentration stable over a long period. It could be argued that the ability to compensate deficits results in altered neuronal activity. A hypothesis that potentially reconciles our results is that the high cerebral activation level in ADHD revealed in fMRI is necessary to compensate inattention and build up concentration. This high activation level for such an easy task, in contrast to the control group, demonstrates a very ineffective way of building up attention and might be understood as the reason for the inability to concentrate for a longer period. Other studies suggest a role of the left parietal cortex, in particular the supramarginal gyrus, in motor attention in healthy population [32]. The findings of activation of parietal regions in ADHD children and adolescents are inconsistent in different studies. Tamm et al. found reduced activity in parietal regions during an oddball paradigm in ADHD [31], whereas Durston et al. found increased activation in the right inferior parietal lobe [4]. In adult ADHD Hale et al. found an increased and decreased activity of parietal regions depending on the complexity of the task in a fMRI study. They conclude that already during relatively basic mental operation (like our task), ADHD subjects use alternative right hemispheric associated visual/spatial compensatory strategies that are less effective during more complex tasks. This might be the reason for the parietal and occipital activation found in our study. According to this idea, we could argue that adults have learned to deal with their symptoms over the course of their lives. Therefore, the different activation patterns could be influenced by the results of a learning process. In respect to the major result of our study that there is evidence for neural compensation in ADHD because of parietal hyperactivation in further investigations we will examine the influence of treatment with methylphenidate on the hypothesized compensatory neural activity.

Notes

Conflicts of interest

None declared.

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