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Differences and similarities between the brains of children with attention deficit hyperactivity disorder and children with autism spectrum disorder

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An analysis of 700 anatomical MRI scans

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Abstract

A strong symptomatic overlap exists between attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD). Yet, no study was so far able to describe the neuroanatomical differences and similarities between the two disorders. To assess the differences existing between the two neurodevelopmental disorders and the features distinguishing abnormal from normal brain, the present study investigated brains of 700 children aged between 8 and 18 years. The data was gathered from the two online databases ABIDE and ADHD-200 and consisted of 173 children diagnosed with ADHD, 115 children with ASD and 412 typically developing children (TDC). By using FreeSurfer's automated segmentation, parcellation and reconstruction technique, information on cortical volume, thickness and surface area, as well as subcortical volume information, has been computed. Results show that children with either disorder have decreased cortical volume in the cingulate gyrus, increased thickness in the frontal lobe and decreased surface area in the occipital lobe. Further, a decrease in thickness, surface area and volume in the temporal lobe were characteristics of ADHD, but not of ASD. The two disorders mostly showed discrepancies in areas of the frontal lobe, around the central sulcus, in subcortical regions (e.g. nucleus accumbens, amygdala, caudate nucleus, and thalamus), in total gray matter volume and in mean cortical thickness. While a postulated general decrease in cortical volume in ADHD could be found, a postulated general cortical increased volume in ASD was not observable. In contrast to previous findings, children with ADHD showed an increased corpus callosum volume, while children with ASD showed no volume abnormalities in this area. This study enlightens the many overlaps and differences in structural brain abnormalities in ADHD and ASD. The findings support the theory of abnormal growth trajectory in many cortical areas. This trajectory seems to eventually converge with the one of normal children in the case of ASD, but seems to remain abnormal in ADHD.

Zusammenfassung

Zwischen der Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung (ADHD) und der Autismus-Spektrum-Störung (ASD) bestehen grosse symptomatische Überschneidungen. Dennoch ist es bisher keiner Studie gelungen, die neuroanatomischen Unterschiede und Gemeinsamkeiten zwischen den beiden Störungen zu erklären. In der vorliegenden Studie wurden die Gehirne von 700 Kindern im Alter von 8 bis 18 Jahren untersucht, um die existierenden Unterschiede zwischen den beiden neuronalen Entwicklungsstörungen festzuhalten, bzw. um zu untersuchen, inwiefern sich anormale Gehirne von normalen Gehirnen unterscheiden. Die verwendeten Daten stammen aus den beiden Online-Datenbanken ABIDE und ADHD-200 und beinhalten die Gehirne von 173 Kindern mit diagnostiziertem ADHD, 115 Kindern mit diagnostiziertem ASD und 412 normal entwickelten Kindern (TDC). Mittels FreeSurfer's automatisierter Segmentierungs-, Parzellierungs- und Rekonstruktions-Technik wurden Parameter zum kortikalen Volumen, zur kortikalen Dicke und Oberfläche, sowie zum subkortikalen Volumen berechnet. Die Ergebnisse zeigen, dass sowohl Kinder mit ADHD und ASD ein verringertes Volumen im ‚cingulären Gyrus, eine erhöhte Dicke im Frontallappen und eine verringerte Oberfläche im Okzipitallappen haben. Als charakteristisch für ADHD, nicht aber für ASD, erwiesen sich eine Verringerung der kortikalen Dicke, der Oberfläche und des Volumens des Temporallappens. Die beiden Störungsbilder zeigten die grössten Unterschiede in Bereichen des Frontallappens, des Sulcus centralis, in subkortikalen Arealen (z.B. Nucleus accumbens, Amygdala, Nucleus caudatus, Thalamus), im Gesamtvolumen der grauen Substanz und der durchschnittlichen kortikalen Dicke. Ein postuliertes verringertes kortikales Volumen konnte in ADHD repliziert werden, während ein postuliertes erhöhtes kortikales Volumen in ASD nicht nachgewiesen werden konnte. Im Gegensatz zu früheren Befunden zeigten Kinder mit ADHD ein erhöhtes Volumen des Corpus callosum. Kinder mit ASD hatten in dieser Region keine Auffälligkeiten. Der vorliegenden Studie gelang es, viele Gemeinsamkeiten und Unterschiede bezüglich strukturellen Auffälligkeiten in ADHD und ASD aufzuzeigen, und sie unterstützt somit die Theorie, dass beide Störungen in diversen kortikalen Bereichen einen anormalen Wachstumsverlauf aufweisen. Im Falle von ASD scheint sich dieser Verlauf schliesslich jenem von normalen Kindern anzugleichen, während dies bei ADHD nicht zutrifft.

Contents

1. Introduction

A child faces many challenges during its development into a healthy adult. Beside many environmental and social obstacles, there are also a lot of neurological causes that can change the path of normal development. Among them are the most common neurodevelopmental disorders, called attention deficit hyperactivity disorder (ADHD) (Castellanos & Proal, 2012) and autism spectrum disorder (ASD) (Amaral, Schumann & Nordahl, 2008). Even though both disorders have a huge impact on the life of affected children and their families, still little is known about the neurological causes, development and effects of ADHD and ASD. One topic currently raising a lot of questions and few answers is the issue of the overlapping symptoms and comorbidities of the two disorders. For instance, children with either disorder show to have attention deficits, overactivity, behavior problems and difficulty with social skills, which complicates a differential diagnosis (Mayes, Calhoun, Mayes & Molitoris, 2012).

The current models are able to explain the main features of the two disorders, when considered as separate (Gargaro, Rinehart, Bradshaw, Tonge & Sheppard, 2011), but struggle to explain the huge overlap of symptoms and comorbidities between ADHD and ASD (Mayes et al., 2012). Thus, newer models suggest that ADHD and ASD may not be as different as believed and should not be regarded as two completely distinguished disorders (Gargaro et al., 2011; Sinzig, Walter & Doepfner, 2009). To confirm these claims and investigate possible neurological correlates that connect or distinguish both disorders, this study analyses the brain structure of children with ADHD and children with ASD, and compares them with one another, as well as with a group of typically developing children (TDC).

To provide with some theoretical background, both disorders will first be introduced separately. Further, some findings showing the possible undistinguishable characteristics of these disorders will be presented. Neurological correlates will be shown and regions of interests (ROI) defined. Relevant hypotheses will be deducted from the given knowledge of literature and finally tested by the available imaging data.

1.1 Attention deficit hyperactivity disorder (ADHD)

ADHD is one of the most common neurodevelopmental disorders known in children with an estimated worldwide prevalence of 5.29% (Polanczyk, de Lima, Horta, Biederman &

Rohde, 2007). This number can increase to 9.5% if a more selected population, such as school-age children in the USA, is studied (CDC, 2010).

According to the DSM-IV-TR and DSM-V, the main symptoms of ADHD are inattention, hyperactivity and impulsivity. Depending on the number of symptoms occurring, ADHD can be divided into three subtypes, called predominantly inattentive type (ADHD-I), predominantly hyperactive-impulsive type (ADHD-H) and combined type (ADHD-C). The diagnosis only holds true if the symptoms cannot be better explained by a pervasive developmental disorder (such as ASD), mood disorder, anxiety disorder, dissociative disorder or personality disorder. While the symptoms had to occur before the age of 7 years according to the DSM-IV-TR, they have to unveil before the age of 12 according to DSM-V (APA, 2000; APA, 2013). Further, it must be stressed that those subtypes of ADHD are not supported by the ICD-10 (WHO, 1992).

ADHD is diagnosed more frequently in males than in females with an estimated male-female ratio of 3:1 (Levi, Hay, Bennett & McStephen, 2005). But this ratio depends on the ADHD subtype as it was found to be 1.7:1 for children with ADHD-H and 4.6:1 for children with ADHD-C (Graetz, Sawyer, Hazell, Arney & Baghurst, 2001).

Moreover, in 50-80% of cases, the disorder persists into adulthood (Frodl & Skokauskas, 2012), where a shift of symptoms occurs towards problems of emotion regulation, disorganization and stress intolerance (Carroll & Rounsaville, 1993; Greydanus, Pratt & Patel, 2007).

1.2 Autism spectrum disorder (ASD)

ASD, as defined by the DSM-V, is a neurodevelopment disorder that incorporates autistic disorder, Asperger's disorder, childhood disintegrative disorder and the diagnosis of pervasive developmental disorder not otherwise specified (PDD-NOS) (APA, 2013). In the previous DSM-IV-TR, these disorders were separated into unique diagnoses, but recent findings suggest that such clear distinction might not be applicable. In their study, Mayes et al. (2012) report that most children with clinical diagnosis of Asperger's disorder actually meet DSM-IV criteria for autism. They further report studies suggesting that the two disorders might differ only in symptom severity or intelligence quotient (IQ). Some studies also divide autism into high (HFA) and low functioning autism (LFA) (Kwon, Ow, Pedatella, Lotspeich & Reiss,

2004). As proposed by Mayes et al. (2012), the study at hand will not make this differentiation, as they both are on the spectrum defined by ASD. Furthermore, HFA is assumed to be similar to Asperger's disorder and the categorization into LFA and HFA isn't supported by DSM-IV (APA, 2000) or ICD-10 (WHO, 1992). For these reasons, this current study will use the term ASD to name the main disorder and refer to the autistic and Asperger's disorder as subgroups of ASD.

The prevalence of ASD ranks between 0.75% and 2.64% and the male-female ratio oscillates between 2.5:1 and 5.1:1, both depending on the studied population of children (Kim et al., 2011).

According to the DSM-V children with ASD show persistent deficits in social communication and social interaction across multiple contexts, as well as restricted, repetitive patterns of behavior, interests, or activities. As implied by the name, the severity of the disorder is set on a spectrum of three levels. The level depends on the degree of social communication impairments and restricted, repetitive patterns of behavior. It ranges from level 1 "requiring support" to level 3 "requiring very substantial support". The diagnosis holds true if the symptoms are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. To be diagnosed with ASD, the symptoms have to be present in the early developmental period of childhood (APA, 2013). The DSM-IV-TR was a bit stricter on this onset and stated that, for the autistic disorder, the symptoms had to occur before the age of 3 years (APA, 2000).

ASD is an enduring condition that generally persists into adulthood (Seltzer, Krauss, Shattuck, Orsmond, Swe & Lord, 2003). Cases where the symptoms seem to disappear occur and might be explained by elaborate behavioral strategies learned by individuals with appropriate preconditions (Helt, Kelley, Kinsbourne, Pandey, Boorstein, Herbert & Fein, 2008)

1.3 Similarities and differences between ADHD and ASD

ADHD and ASD are both neurodevelopmental disorders that affect a lot of children worldwide but based solely on their diagnostic descriptions; ADHD and ASD seem to have little in common. The ICD-10 and DSM-IV-TR even preclude a dual-diagnosis of the two disorders. In other words, the diagnosis for ADHD should be changed to autistic disorder if the symptoms are better suited by autism (APA, 2000; Gargaro et al., 2011; WHO, 1993).

This preclusive view on both disorders is rather unsatisfying, as evidence suggest a lot of symptomatic overlapping and co-occurrence of both disorders (Geurts, Grasman, Verté, Oosterlaan, Roeyers, van Kammen & Sergeant, 2008). Fortunately, in the newer DSM-V, ASD is no longer listed as an exclusion criterion for ADHD (APA, 2013). This is improvement, as it is more and more accepted that ADHD symptoms are common in children with ASD and that both disorders have many similar comorbidities. But the question to which degree ASD symptoms are common in children with ADHD remains open (Mayes et al., 2012, Geurts et al., 2008).

In the following two sections, distinction between the two disorders will be clarified by emphasizing the behavioral and cognitive similarities and differences between ADHD and ASD.

1.3.1 Behavioral and cognitive similarities

Additionally to the previously mentioned similarities between ADHD and ASD, such as being neurodevelopmental disorders with preponderance in males (Fombonne, 2003), both groups show similar neuropsychological deficits. A good summary of those deficits was realized by Mayes et al. (2012, p. 277-278) who report that, in comparison to the norm, children with ADHD and ASD both show more irritability, more anger, have more behavioral problems, show similar neurocognitive weaknesses, including deficits in executive functions, slow processing speed, dysgraphia, learning disability in written expression, deficits in attention, motor control, and perception and often have early language delay and sleep problems. ADHD as well as ASD show high comorbidity to fronto-striatal disorders such as obsessive-compulsive disorder (OCD) and Tourette's disorder (Gargaro et al., 2011; Geurts et al., 2008).

Many studies showed that inattention, impulsivity and hyperactivity, the core symptoms of ADHD, are quite common among children with ASD (Brieber et al., 2007; Gargaro et al., 2011; Mayes et al., 2012). Simonoff, Pickles, Charman, Chandler, Loucas & Baird (2008) found that ADHD is actually the second most common comorbid disorder of ASD. As reviewed by Brieber et al. (2007, p. 1251) "patients with ADHD also show problems in social interaction and communication, albeit a smaller degree than patients with ASD." Therefore it is not surprising that Rommelse, Franke, Geurts, Hartman & Buitelaar (2010) found that 20-50% of children with ADHD also meet the criteria for ASD and 30-80% of children with ASD meet the criteria for ADHD as well. This high overlap between the two disorders often

leads to a delayed diagnosis of autism or even to misdiagnoses as ADHD (Hartley & Sikora, 2009).

All those comorbidities and similarities between the two disorders suggest an underlying common factor between ADHD and ASD. Thus, the study at hand aims at investigating similarities and co-occurrence between the disorders, at the level of the brain structure.

1.3.2 Behavioral and cognitive differences

Despite the similarities between ADHD and ASD mentioned above, there are also a lot of features that differentiate the two disorders from each other. As reviewed by Mayes et al. (2012, p. 278), "children with autism had more problems with language and communication, social interaction, peer relationships, stereotyped and idiosyncratic language, and imaginative play, relating to people, emotional responsiveness, stereotypies, odd and repetitive object use, eye contact, too much or too little fear, and verbal and nonverbal communication." In contrast, children with ADHD show to have greater difficulty with motor inhibition tasks than children with autism (Mahone, Powell, Loftis, Goldberg, Denckla & Mostofsky, 2006). Mayes et al. (2012) found that children with autism have a significantly higher frequency of selective attention than children with ADHD-C and ADHD-I. This is in line with the fact that children with ASD are well known for their ability to hyperfocus on topics they are interested in. In contrast to children with ADHD, children with autism did not show difficulties on a working memory task (Geurts, Verté, Oosterlaan, Roeyers & Sergeant, 2004). This holds true even for autistic children with additional symptoms of ADHD (Gargaro et al., 2011).

As seen above, both disorders show deficits in executive functions. Executive functions are mental control processes, e.g. response inhibition, working memory, cognitive flexibility (set shifting), planning, and fluency, that enable self-control and are necessary to achieve a future goal by maintaining appropriate problem solving strategies (Geurts et al., 2004). In some executive functions ADHD and ASD are similarly restricted (Barnard-Brak, 2011; Mayes et al., 2012), whereas in others, they seem to show a complementary pattern of deficits. As reviewed by Gargaro et al. (2011, p. 1084), "children with ADHD show difficulties on tasks measuring inhibition and sustained attention while remaining relatively unaffected on tasks measuring planning or cognitive flexibility. Children with autism in contrast, appear to display preservation of conscious inhibitory function, but quite severe problems in planning and shifting attention." Gargaro et al. (2011) even suspect a double dissociation between the

two disorders, if the focus is put on executive functions such as inhibition, planning and flexibility. Geurts et al. (2004) suggest that the deficits in cognitive flexibility in autistic children might be related to the symptom of restricted, repetitive patterns of behavior, and therefore be less frequent in children with ADHD.

In consideration of all these differences between the two disorders, Mayes et al. (2012) tested their Checklist for Autism Spectrum Disorder (CASD) on a sample of 847 children with autism and 158 children with ADHD and found that all children with autism had at least 15 of the 30 symptoms of the CASD. The mean number of symptoms of the ASD group was 22, while the mean of the ADHD group was 4 and typical children's one was 1.3. They found that three symptoms were only present in children with autism (i.e. unusual fascination with repetitive movements, language regression and special abilities relative to other abilities). That is, children having at least one of those three CASD symptoms had autism. Additionally, almost all 30 symptoms were found in over half of the 847 children with autism. Most children with ADHD-I showed none of the 30 symptoms, and only two (i.e. problems with social skills and over reactivity, meltdowns, or aggression) were present in the majority of children with ADHD-C. Therefore the CASD is very reliable on distinguishing children with autism from children with ADHD and from typical children solely on their symptom profiles, with an accuracy of 99.5% and 100% respectively (Mayes et al., 2012).

As demonstrated, many features make a clear cut between ADHD and ASD. Thus, the current work also investigate for differential features in the structure of the brain between the disorders.

1.4 Neurological findings in children with ADHD or ASD

The two disorders can't just be compared on their characteristics in their behavioral and cognitive traits; they also share and differ in their neurological appearance. The purpose of this section is to summarize the chorus of the neurological findings in children of both disorders and therefore show the degree of neurological distinction and overlap between them. As this study only looks at anatomical T1-weighted magnetic resonance imaging (MRI) scans, findings acquired through methods such as functional resting state MRI (rsfMRI) or diffusion tensor imaging (DTI), a noninvasive way of mapping white matter pathways, will be disregarded (For more on those topics see: Alexander et al.2007; Assaf et al., 2010; Barnea-Goraly, Kwon, Menon, Eliez, Lotspeich & Reiss, 2004; Castellanos & Proal, 2012; Courch-

esne & Pierce, 2005; Dickstein, Bannon, Castellanos & Milham, 2006; Konrad & Eickhoff, 2010). For a summary of all results mentioned in this section, see *Table 1*.

1.4.1 Structural abnormalities

This section reports the neuroanatomical findings in studies including children with ADHD or ASD. Due to the extent and heterogeneity in these findings, only regions with the most prominent features are reported. It must be stressed that some studies only showed unilateral findings in some regions. However, since the consensus of regional findings is rather inconsistent in their lateralization, the regions are further listed as non-lateralized, and therefore represent both hemispheres. Moreover, some studies only investigated a sub population of children, e.g. only children with Asperger's or children with ADHD-C, while other studies investigated the either disorder as a whole. These populations were accounted for either the ASD or the ADHD syndrome. In addition, the reader should keep in mind that there is no existent single study that enlightened at once all the abnormalities reported in the following section. That is, even for the regions representing the most prominent findings, there were also studies whose results were not in line with previous results or even showed contradictory findings. Many factors may account for this, such as a low number of subjects, age differences between samples, neglect of relevant covariates (e.g. total brain volume, IQ or age), inclusion or exclusion criteria, the choice of control group or the methods used (Hyde, Samson, Evans & Mottron, 2010)

Therefore the following results have to be taken with precaution, even though they have been thoroughly reviewed and reported with the best possible accuracy for them to be representative of the current knowledge in the field. The findings are structured as follow: total brain volume, frontal, temporal, parietal and occipital lobe, anterior cingulate cortex, corpus callosum, cerebellum, basal ganglia, amygdala, thalamus and hippocampus. Disorder specific findings are reported separately in each section.

TOTAL BRAIN VOLUME

Total brain volume and ASD: The most prominent finding in volume differences between children with ASD and TDC is an increased brain volume in ASD children (Gargaro et al., 2011). As reviewed by Amaral et al. (2008) the brain of children with autism seems to develop normally until the age of 12 months. Later, , growth trajectory is increased in comparison to TDC, leading to a 5-10% increase in total brain volume in children with autism be-

tween the age of 18 months and 4 years. These findings are supported by another meta-analysis done by Redcay & Courchesne (2005), who found that children with autism show the greatest deviation of total brain volume from normal developing cortical volume between the age of 2 and 5 years. They finally reach a plateau, leading their total brain volume to rank within the range of TDC. It is important to mention that some of the measurements for total brain volume in very young children (i.e. up to 3 years old), are only based on measuring the head circumference, which was reported to be a good proxy for brain size in young age (Amaral et al., 2008; Courchesne et al., 2001). The difference of total brain volume is not solely driven by increased gray matter volume. Indeed, Courchesne et al. (2001),found that autistic children of 2 to 3 years old had a 18% increase in white matter volume in the cerebrum, whilst having only 12% increased cerebral cortical gray matter volume. Similar results were reported by Carper, Moses, Tigue & Courchesne (2002) who found up to 20% enlarged gray and white matter volume in 2 to 3 years old children with autism. In their data, the authors hypothesize an anterior to posterior gradient of overgrowth, as the frontal lobe showed the greatest enlargement while the occipital lobe was not significantly different from TDC. However, Carper et al. (2002) found no gray or white matter differences between older children with autism compared with TDC. Only few studies focused on mean cortical thickness, and their findings are rather inconsistent. A longitudinal study conducted by Schumann et al. (2010) also revealed that all regions, but the occipital lobe, showed an abnormal increased growth rate. Further, Hardan, Muddasani, Vemulapalli, Keshavan & Minshew (2006b) showed that autistic children of 8 to 12 years had an increased mean cortical thickness, which was primarily driven by the parietal and temporal lobe, as no significant thickness difference was found in frontal and occipital lobe. In contrast, a study conducted in adults with autism found regions with decreased thickness in the frontal, parietal and temporal lobe (Hadjikhani, Joseph, Snyder & Tager-Flusberg, 2006).

Total brain volume and ADHD: The most prominent finding in volume differences between children with ADHD and TDC is a total brain volume reduction (Valera, Faraone, Murray & Seidman, 2007). The amount of decreased total brain volume is reported to be between 3.2% (Castellanos et al., 2002a) and 5.4% (Carmona et al., 2005). In their longitudinal study investigating children with ADHD from the age of 5 to 18 years, Castellanos et al. (2002a) reported that reduced global brain volume persists into adulthood. It remains unclear whether total brain volume differences between children with and without ADHD are driven by gray and/or white matter differences. Indeed, some studies found no significant reduction in global white matter volume (Carmona et al., 2005; Jiao, Chen, Ke, Chu, Lu & Herskovits,

2010), while others found up to 10.7% white matter volume reduction (Castellanos et al., 2002a). In another longitudinal study, children with ADHD were measured between the age of 8.7 and 15.3 years, Shaw et al. (2006) found that children with ADHD had a significantly reduced mean cortical thickness, driven by significant reduction in superior/medial prefrontal, right anterior/mesial temporal and left precentral located clusters. Interestingly, children with worse clinical outcomes showed greater decrease in cortical thickness in the left medial prefrontal cortex (vs. better outcome group or control group at baseline). These findings are supported by a study of Narr et al. (2009), who found that ADHD children between the age of 7.2 and 16 years globally showed significant reduction in cortical thickness. Further, Shaw et al. (2007) found that the growth of the cortical thickness is disturbed in children with ADHD, which could account for their outcome. While 50% of the TDC reached the thickness peak throughout most of the cortex by the age of 7.5 years, 50 % of children with ADHD reached the thickness peak by 10.5 years old. This significant delay was reported to be most prominent in prefrontal regions, but was also observable in the middle and superior temporal cortex, as well as in the middle occipital gyrus. In contrast, the results of Wolosin, Richardson, Hennessey, Denckla & Mostofsky (2009) found no cortical thickness abnormalities in 8.7 to 12.8 years old children with ADHD. But these authors found decreased surface area in bilaterally frontal, right temporal, right parietal and left occipital regions.

FRONTAL LOBE

The frontal lobe can be sub-divided into five main regions, which are the orbital, dorsolateral, mesial prefrontal, premotor, and motor regions. Lesion studies have shown that the orbital frontal region is associated with social disinhibition and impulse control, and that the dorsolateral prefrontal cortex (DLPFC) is associated with executive functions such as planning, working memory, and attention. Mesial prefrontal regions are associated with word disfluency and slowing down of spontaneous behaviors. The motor and premotor regions are associated with motor movements (Seidman, Valera & Makris, 2005).

Frontal lobe and ASD: As reviewed by Amaral et al. (2008), there is mounting evidence for increased volume in frontal lobe of ASD children when compared to TDC. The authors further report that these volume enlargements do not seem to be specific to certain subregions of the frontal lobe. Nonetheless, they mention possible increased volume in the dorsolateral prefrontal, the medial frontal and in the right orbitofrontal cortex. In a study that investigated cortical thickness, Jiao et al. (2010) found that children with ASD showed decreased

cortical thickness bilaterally in the pars triangularis (a part of the inferior frontal gyrus), in the left medial orbitofrontal gyrus and in the left frontal pole compared to control subjects.

Frontal lobe and ADHD: In their meta-analysis, Seidman et al. (2005) report that children with ADHD show most prominent volume reduction in frontal lobe, in DLPFC. However, they also report studies showing reduced volume in the right superior frontal gyrus, as well as a reduced surface area bilaterally in inferior portions of the DLPFC. These findings are in line with a voxel-based morphometry (VBM) study conducted by Carmona et al. (2005), who found that ADHD children aged of 6 to 16 years had decreased volume in left DLPFC, in orbitofrontal cortex, as well as in left fronto-parietal areas of the motor, premotor and somatosensory cortex.

TEMPORAL LOBE

The temporal lobe is, amongst other, associated with auditory and linguistic relevant functions and skills that are impaired in children with ASD or ADHD (Mayes et al., 2012; Seidman et al., 2005).

Brieber et al. (2007) found that, in comparison to TDC, children with ASD showed reduced gray matter volume bilaterally in the inferior temporal gyrus, while children with ADHD only showed this reduction in the left superior temporal gyrus. Those results are supported by a study conducted by Kwon et al. (2004) who found decreased gray matter density in the ventromedial regions of the temporal cortex in 10 to 18 years old males with ASD. Volume reduction in the temporal lobe in ADHD children is also supported by the studies of Carmona et al. (2005) and Castellanos et al. (2002a). Sowell, Thompson, Welcome, Henkenius, Toga & Peterson (2003) found that children with ADHD aged of 8 to 16 years had reduced gray matter density bilaterally in the lateral aspects of anterior and mid-temporal cortices and increased gray matter density in the posterior temporal lobe.

Information about cortical thickness abnormalities in ASD is rather inconsistent. While Hardan et al. (2006b) found the mean cortical thickness and the thickness in the temporal lobe to be increased, Wallace, Dankner, Kenworthy, Giedd & Martin (2010) found a thinner temporal lobe in ASD children and adolescents aged of 12 to 24 years. A thinner cortex is supported by a longitudinal study of Hardan, Libove, Keshavan, Melhem & Minshew (2009), who measured children between 8 and 12 years old with an interval of 30 months between the scans. They found that children with ASD had significant cortical thinning over the whole cortex, while the cortical thinning in the control group was restricted to the parietal lobe.

PARIETAL LOBE

Brieber et al. (2007) investigated volume differences in children with ASD, ADHD or TDC, and found that the gray matter volume was increased in children with ASD in the right supramarginal gyrus, as well as in the left postcentral gyrus, and that the gray matter volume in children with ADHD was increased in the bilateral superior parietal and postcentral gyrus. A conjunction analysis conducted on this data revealed that the volume in the left inferior parietal gyrus and postcentral gyrus was increased in both disorders. In 8 to 18 years old children with ADHD, Sowell et al. (2003) found increased gray matter density in the inferior parietal lobe. In contrast, Carmona et al. (2005) found reduced gray matter volume in the precuneus of children with ADHD. Castellanos et al. (2002a) found up to a 4.1% reduction of parietal gray matter volume, but only if the data were not corrected for multiple comparisons.

A study with ASD children found increased cortical thickness in the left precuneus, a part of the superior parietal lobe (Jiao et al., 2010). This stands in contrast to the findings of Wallace et al. (2010), who reported cortical thinning in the parietal lobe in children and adults with ASD between the age of 12 and 24 years, even after correcting for IQ and medication.

OCCIPITAL LOBE

Only few studies report volume differences between ASD or ADHD children with TDC. In a study that focused on children with ASD or ADHD, Brieber et al. (2007) found that the gray matter volume in the left middle occipital gyrus was significantly reduced for both disorders compared with TDC. The decreased occipital volume in children with ADHD is further supported by many studies and was mostly reported to be present in the left hemisphere only (Castellanos et al., 2002a; Durston, Pol, Schnack, Buitelaar, Steenhuis, Minderaa & Kahn, 2004; McAlonan et al., 2007).

ANTERIOR CINGULATE CORTEX

The anterior cingulate cortex (ACC) lies just above the corpus callosum. The dorsal part of the ACC has strong connections to the DLPFC and seems to be involved in cognitive processes such as target detection, response selection, error detection, and reward-based decision making, all functions thought to be impaired in ADHD (Seidman et al., 2005).

ACC and ASD: To my knowledge, there are no findings in the literature reporting volume abnormalities in the ACC between children with ASD and TDC. Solely one study focusing on cortical thickness found increased cortical thickness in the left caudal ACC in children with ASD between the age of 6 and 15 years, compared to their controls (Jiao et al., 2010).

ACC and ADHD: In their meta-analysis Frodl & Skokauskas (2012) suggest that the ACC volume of untreated ADHD children is reduced compared with TDC. Carmona et al., (2005) found that the gray matter volume in the whole cingulate cortex (anterior, middle and posterior) was decreased in the left hemisphere of 6 to 16 years old children with ADHD, compared to their controls. A speculation of a connection between children with ADHD and volume changes in the ACC is supported by another study that found significantly smaller gray matter volumes bilaterally in the ACC of adults with ADHD, in comparison to healthy adults (Amico, Stauber, Koutsouleris & Frodl, 2011).

CORPUS CALLOSUM

The role of the corpus callosum (CC) is rather simple, but nonetheless essential. As the largest pathway in the human brain, this fiber tract is responsible for the main transfer of information between the two hemispheres (Sokol & Edwards-Brown, 2004). The CC is topologically organized, meaning that the anterior CC connects prefrontal homologues and heterologous cortical areas, the middle CC connects the premotor, supplementary motor and motor areas, and that the posterior CC connects the parietal, temporal and occipital cortical areas (Hofer & Frahm, 2006; Hutchinson, Mathias & Banich, 2008).

Corpus callosum and ASD: A meta-analysis that investigated a total of 253 patients with autism (mean age = 14.58 years, SD = 6.00 years) and 250 healthy control subjects (mean age = 14.47 years, SD = 5.31 years) found that the total area of the CC was significantly reduced in autism and that this reduction was greatest in the anterior part, being less prominent more caudally (Frazier & Hardan, 2009). Those results are supported by findings that adults with autism have a reduced anterior CC volume, even after correcting for total brain size (Hardan, Minshew & Keshavan, 2000). Piven, Bailey, Ranson & Arndt (1997) also found a reduced CC volume in a population of autistic adults, but only in the body (middle) and posterior part of it.

Corpus callosum and ADHD: Many studies found that children with ADHD have reduced CC volume compared to TDC (as reviewed by Gargaro et al., 2011; Hutchinson et al., 2008; Valera et al., 2007). In their meta-analysis, Hutchinson et al. (2008) found that the effect of reduced volume seems to be most prominent in the splenium, i.e. the most posterior part of CC. This finding is in line with results in children with ADHD aged of 7 to 12 years, who showed smaller total CC and splenium area than TDC (Hill, Yeo, Campbell, Hart, Vigil & Brooks, 2003).

CEREBELLUM

The cerebellum was traditionally associated with coordination and execution of motor movements, but recent studies have shown that, through connections with frontal regions, the cerebellum is also involved in cognitive and affective processes such as timing, attentional shifting, executive skills, social behavior, and a flattening of affect (Krain & Castellanos, 2006; Seidman et al., 2005; Sokol & Edwards-Brown, 2004).

Cerebellum and ASD: In general, the volume of the cerebellum in children with autism seems to be increased compared to healthy controls (Amaral et al., 2008, Stanfield, McIntosh, Spencer, Philip, Gaur & Lawrie, 2008). Autistic children aged of 2 to 3 years showed a 39% increased white matter volume in the cerebellum compared to their healthy controls (Courchesne et al., 2001). But this difference was not significant anymore if a cohort of 12 to 16 years old autistic children were considered. It is unclear whether the cerebellar enlargement is region-specific or only represents an overall enlargement, as it is mostly found to be proportional to the total brain volume increase in autism (Amaral et al., 2008).

Cerebellum and ADHD: Many studies showed a significant decrease in cerebellar volume in children with ADHD compared to healthy controls (Carmona et al., 2005; Castellanos & Tannock, 2002b; Kobel et al., 2010). A meta-analysis by Valera et al. (2007) found that reduced cerebellar volume was most pronounced in the posterior inferior cerebellar vermis. As shown in a longitudinal study, reduced cerebellar volume seems to persist into adulthood (Castellanos et al., 2002a).

BASAL GANGLIA

The basal ganglia are composed of the caudate nucleus, the putamen and the globus pallidus. Those regions are associated with a wide array of functions such as motor selection, preparation, and execution, cognitive control, as well as motivational and emotional processes (Di Martino et al., 2008). Sears, Vest, Mohamed, Bailey, Ranson & Piven (1999) found that increased volume in the caudate nucleus correlated positively with complex repetitive motor behavior and negatively with compulsive and ritual behaviors. It is suspected that a disruption of the frontal-striatal pathway may underlie the stereotyped behavior in autism (Sears et al. 1999).

Basal ganglia and ASD: There is little to none evidence of volume difference in the basal ganglia between children with ASD and TDC (Amaral et al., 2008). Langen, Durston, Staal, Palmen & van Engeland (2007) found an increased caudate volume in a sample of autistic subjects with an age range of 15.5 to 24.7 years, even after the correction of total brain

volume. However, this was only significant in a sample of 6.9 to 14.6 years old autistic children, if the correction for whole brain volume was not applied. In a sample of adults with autism, Hollander et al. (2005) found increased right caudate volume, even after controlling for total brain volume. This is not in line with the study of Sears et al. (1999), who found that the effect of increased caudate volume in a group of 12 to 29 years old autistic subjects was no longer present when total brain volume was accounted for.

Basal ganglia and ADHD:

In contrast to ASD, volume differences in the basal ganglia are quite more prominent in children with ADHD compared to TDC. Many studies have found that the caudate nucleus, the putamen, the globus pallidus (i.e. the basal ganglia in general) show significant reduced volumes in children with ADHD compared to TDC (Ellison-Wright, Ellison-Wright & Bullmore, 2008; Frodl & Skokauskas, 2012; Valera et al., 2007). In their longitudinal study, Castellanos et al. (2002a) found that initial differences in caudate nucleus volume disappeared with age, as the caudate volume of TDC decreased to the level of children with ADHD. It must be stressed that some findings of reduced volume in the basal ganglia were only found in ADHD boys (vs. girls) (Qiu, Crocetti, Adler, Mahone, Denckla, Miller & Mostofsky, 2009) or were not significant anymore when gender was considered as covariate (Frodl & Skokauskas, 2012). This suggests that gender has to be considered when studying volume changes in children with ADHD.

AMYGDALA

The amygdala is a subcortical region below the temporal lobe. It is most prominently known to be involved in fear and anxiety processing and in emotional functions such as empathy, theory of mind and other social cognitions (Stanfield et al., 2008).

Amygdala and ASD: Sparks et al. (2002) found that, in comparison to TDC, 36-56 months old children with autism had an increased amygdala volume (13.64% left and 16.67% right). This finding is supported by a longitudinal study on children with ADHD aged of 2 to 4 years, showing not only an increased amygdalar volume, but also a significantly increased amygdalar growth rate (Nordahl, Scholz, Yang, Buonocore, Simon, Rogers & Amaral, 2012). Schumann et al. (2004) found the same results in children with autism aged of 7.5 to 12.5 years, but mentioned that the enlargement of the amygdala was no more detectable when an older group of children aged of 12.75 to 18.5 years was considered. Schumann et al. (2004) suspect that the developmental growth trajectory of the amygdala rather than its resulting volume is different in children with autism in comparison with TDC. That is, in children with

autism, the growth rate of the amygdala is steeper under the age of 12 years, leading to a larger amygdalar volume in children with ASD than in TDC. However, this growth rate is flatter after the age of 12 years, until there is no more amygdala volume difference detectable in adolescent.

Amygdala and ADHD:

There are no consistent findings concerning volume differences in the amygdala in children with ADHD in comparison to TDC. However, a surface analysis in children aged of 6 to 18 years suggested that the surface area of the amygdala is bilaterally reduced in children with ADHD. That is, there are differences in the shape of the amygdala. Those differences were located primarily over the basal nucleus of the right and lateral nucleus of the left amygdala (Plessen et al., 2006). Further, studies comparing adults with ADHD to healthy controls show inconsistent findings. In a population of 19 to 55 years old adults with ADHD, Perlov et al. (2008) found no significant differences in the amygdalar volume, while Frodl et al. (2010) found significantly smaller bilateral amygdalar volumes in adults with ADHD with a mean age of 33.6 years. Reduced volume in the right amygdala was correlated with the occurrence of more hyperactivity and less inattention (Frodl et al., 2010).

THALAMUS

The thalamus is a very important brain structure in the middle of our brain, responsible for relaying the informational flow into our brain and to the relevant cortical areas. The cortico-thalamo-cortical pathways are essential for ongoing cortical processing (Sherman, 2006).

There is only little to none evidence that the thalamic volume is different in children with ADHD (Xia, Li, Kimball, Kelly, Lesser & Branch, 2012) or children with ASD (Amaral et al., 2008) compared to TDC. There are two studies showing that autistic people between the age of 8 and 45 years have no significant correlation between the thalamic volume and the total brain volume (Tsatsanis, Rourke, Klin, Volkmar, Cicchetti & Schultz, 2003; Hardan, Girgis, Adams, Gilbert, Keshavan & Minshew, 2006a). Normally, a positive correlation between an increase in thalamic volume and an increase in total brain volume can be found in TDC, and the lack of such correlation is sometimes explained by a possible underdevelopment of connections between cortical and subcortical regions, which in turn could explain a decreased thalamic volume.

In their population of 9 to 15 years old children with ADHD, Xia et al. (2012) found reduced thalamic volume in children with ADHD compared with TDC, but only in the right

hemisphere. As the results were not controlled for total brain volume, it is hard to say if this thalamic volume difference is driven by a local or a global change in brain size.

HIPPOCAMPUS

The hippocampus is essential for memory and learning (Williams & Minshew 2007), as well as for other cognitive functions including processing of spatial information (Maguire, Gadian, Johnsrude, Good, Ashburner, Frackowiak & Frith, 2000).

Hippocampus and ASD: In children aged of 36-56 months, Sparks et al. (2002) found that, in comparison to TDC, children with autism had an increased hippocampal volume (8.54% left and 9.16% right). These findings are supported by another study showing that autistic children from the age of 7.5 to 18.5 years had bilateral increased hippocampal volume in comparison to TDC (Schumann et al., 2004).

Hippocampus and ADHD: Plessen et al. (2006) found that, in comparison to TDC, a group of children with ADHD between the age of 6 and 18 years had bilaterally increased hippocampus volume and a corresponding surface analysis suggested that this difference in volume was mainly driven by an enlarged anterior hippocampus. An analysis in adults showed no such hippocampal volume difference between people with and without ADHD (Perlov et al., 2008; Frodl et al., 2010).

1.4.2 Summary

All previously mentioned neurological findings distinguishing people with ADHD from TDC or ASD from TDC are summarized in the following *Table 1* and *Table 2*. This summary should help the reader to consolidate the previous reports and gives me the opportunity to point to common trends and inconsistencies in the findings.

Table 1 *Summary table of previous mentioned neurological findings in children with ADHD or ASD in comparison to TDC*

Volume	***ASD***	***ADHD***
Cerebral Cortex	Increased	Decreased
Frontal lobe	Increased	Decreased
Superior frontal gyrus		Decreased
Orbitofrontal cortex	Increased	Decreased
Dorsolateral prefrontal cortex	Increased	Decreased
Medial frontal cortex	Increased	
Motor cortex		Decreased
Premotor cortex		Decreased
Temporal lobe	Decreased	Decreased
Superior temporal gyrus		Decreased
Anterior and mid-temporal		Decreased
Posterior temporal lobe		Increased
Inferior temporal gyrus	Decreased	
Parietal lobe	Increased	Inconsistent: Increased or Decreased
Postcentral gyrus	Increased	Increased
Somatosensory cortex		Decreased
Inferior parietal gyrus	Increased	Increased
Supramarginal gyrus	Increased	
Superior parietal gyrus		Increased
Precuneus		Decreased
Occipital lobe	Decreased or no differences	Decreased
Middle occipital gyrus	Decreased	Decreased
Anterior Cingulate Cortex		Decreased
Corpus callosum	Decreased	Decreased
Cerebellum	Increased	Decreased
Subcortical Structures		
Basal ganglia		Decreased
Caudate Nucleus	Increased	Decreased
Putamen		Decreased
Globus Pallidus		Decreased
Amygdala	Increased	Decreased (in adults)
Thalamus	Decreased	Decreased
Hippocampus	Increased	Increased

Thickness	***ASD***	***ADHD***
Cerebral Cortex	Increased between 8 to 12 years and decreased in adulthood	Decreased
Frontal lobe	Decreased in pars triangularis, medial orbitofrontal gyrus and frontal pole	
Prefrontal Cortex		Decreased
Precentral gyrus (left)		Decreased
Temporal lobe	Inconsistent: Increased or Decreased	Decreased
Parietal lobe	Decreased	
Precuneus	Increased	
Anterior Cingulate Cortex (left)	Increased	

Surface Area	***ASD***	***ADHD***
Cerebral Cortex		Decreased
Frontal lobe		Decreased
Dorsolateral prefrontal cortex		Decreased
Temporal lobe (right)		Decreased
Parietal lobe (right)		Decreased
Occipital lobe (left)		Decreased
Corpus callosum	Decreased (effect biggest anterior and decreasing caudally)	Decreased
Subcortical Structures		
Amygdala		Decreased

The terms "increased" or "decreased" represent the direction of the main findings of changes in children with a disorder in contrast to TDC.

It is evident that there is a clear difference in many brain regions between children with ASD and children with ADHD. In general, children with ASD seem to have a larger brain compared to TDC, whereas children with ADHD seem to have a smaller brain compared to TDC. But despite the differences, there are also some regions showing similar abnormalities. For example, both disorders show a decreased volume in mid-sagittal area of the corpus callosum, the temporal lobe and the thalamus, and an increased volume in the hippocampus.

It must be kept in mind that *Table 1* is just an attempt to summarize the rather big and very heterogeneous pool of findings concerning structural abnormalities in ASD or ADHD. Many of these findings can be lateralized, appear only in a specific subpopulation of children or do not account for the influence of confounding variables such as age, IQ or total gray matter volume. Good examples for the inconsistencies in the field are reports about amygdalar and hippocampal volume abnormalities in children with ASD. While some studies reported an increase in amygdalar volume (Howard, Cowell, Boucher, Broks, Mayes, Farrant & Roberts, 2000; Nordahl et al., 2012), others reported a decrease (Aylward et al., 1999) or failed to find any abnormalities at all (Haznedar, Buchsbaum, Wei, Hof, Cartwright, Bienstock & Holland-

er, 2000). Similarly, some studies reported a volume increase in the hippocampus (Schumann et al., 2004; Sparks et al., 2002), while others reported a decrease (Aylward et al., 1999) or no differences between ASD and TDC (Piven, Bailey, Ranson & Arndt, 1998). More discrepancies in ASD findings can be found in the paper by Hyde et al. (2010), and a good review about discrepancies in ADHD findings is provided by Baumeister & Hawkins (2001).

Table 2 *Summary of abnormal growth trajectories in children with ASD or ADHD*

Growth Trajectory	*ASD*	*ADHD*
Total cerebral volume	Increase growth after 1 year with a peak around 4 years and normal volume by adulthood	Decreased volume consists into adulthood
Cerebellum volume	Increased volume with 2-3 years and normal volume with 12 to 16 years	Decreased volume consists into adulthood
Amygdala volume	Increased growth before 12 years, decreased growth after 12 years and normal volume in adulthood	
Caudate Nucleus volume		Decreased volume with 2-3 years and normal volume with 12 to 18 years
Hippocampus volume		Decreased volume as child does not consist into adulthood
Cerebral thickness		3 year delayed peak thickness at age of 10.5 years

It must be noticed that some of the brain abnormalities in children with either ADHD or ASD seem to disappear when the children become older. The fact that brain abnormalities in ASD, such as total cerebral, cerebellar and amygdalar volume differences, seem to fade away in adulthood suggest that ASD might rather be a disorder of disturbed growth trajectory than a disorder of abnormal absolute growth outcome (Amaral et al., 2008). In contrast, ADHD sees its main volume abnormalities persist into adulthood (Castellanos et al., 2002a). A summary of the suggested abnormal growth trajectories in ASD or ADHD can be found in *Table 2*.

The question of a possible link between the fact that an ASD brain becomes normal and an ADHD one remains smaller, and the fact that ADHD children might recover from the disorder with aging (Frodl & Skokauskas, 2012), whereas children with ASD mostly do not recover (Seltzer, Krauss, Shattuck, Orsmond, Swe & Lord, 2003), remains open.

1.5 Theories, Models and Explanations for ADHD and ASD

Even though a huge consortium of studies tried to explain either ADHD or ASD, there is still no general accepted model that is capable of explaining one of the disorders, let alone both together. Nonetheless, there are many attempts to reach this goal (Gargaro et al., 2009; Sinzig et al., 2009). The aims of this section are first to describe the most prominent theories and models that try to explain the two disorders and second to summarize all these features into one common collective explanation.

1.5.1 The theory of executive dysfunction

The theory of executive dysfunction stresses that ADHD and ASD are both disorders with impaired executive functions, such as planning, working memory, inhibition, impulse control, initiation, monitoring of action, and sustained attention (Barnard-Brak, 2011; Hill, 2004). As previously mentioned, the two disorders share common weaknesses in executive functions (Mayes et al., 2012; Pennington & Ozonoff, 1996), but differ in the degree of their impairment (Gargaro et al., 2011). These executive dysfunctions are linked to frontal lobe abnormalities, as shown with lesion studies reporting that frontal lobe damage leads to impairment in executive functions (Hill, 2004) and similar behavioral characteristics observed in ADHD and ASD (Geurts et al., 2004). The theory of executive dysfunction is validated by the fact that the frontal lobe is the brain region that last matures (Bradshaw, 2001). Due to its longer development rate, the frontal lobe is more susceptible to get impacted by interferences and is a critical and vulnerable region in neurodevelopmental disorders such as ADHD and ASD (Gargaro et al., 2011). The theory of executive dysfunction is a good approach to explain ADHD and ASD, as many of the fronto-striatal structures being abnormal in children affected by these disorders, such as prefrontal cortex, dorsal anterior cingulate cortex, caudate nucleus and putamen, are part of the so called executive function network (Makris et al., 2007). However, a meta-analysis by Willcutt, Doyle, Nigg, Faraone & Pennington (2005) challenged the validity of this theory. They report that even though weaknesses in executive functions are an important component of ADHD, they cannot account for all cases of the disorder.

1.5.2 The aberrant connectivity theory

The aberrant connectivity theory assumes that the underlying cause of ADHD and ASD is dysfunctional long-distance cortico-cortical connections. Redcay & Courchesne (2005) as-

sume that the rapid growth and abrupt cessation of growth negatively affect the creation of long-distance connections in the brain of children with ASD. They hypothesize that a shorter window of experience-dependent changes negatively influences the development of associative cortices. This would force the ASD child's brain towards a strategy of local processing, rather than to a more coherent, global, multi-sensory and contextual processing. The theory is supported by DTI studies and postmortem findings, and can explain deficits in executive functions, social cognition and language (Frazier & Hardan, 2009), at least for ASD.

In an analog way, the delay of such window of experience-dependent changes could cause similar aberrant functional connectivity in ADHD. The hypothesis of an aberrant connected brain in ADHD is rather new and not yet deeply investigated. Nonetheless, reports from structural and functional connectivity studies suggest that a reduction of long-distance connections could explain the global efficiency deficit found in ADHD (Konrad & Eickhoff, 2010).

This theory is further supported by numerous findings of a dysfunctional fronto-striatal network in ADHD and ASD (Bradshaw, 2001; Ellison-Wright et al., 2008; Gargaro et al., 2011), as well as by reports of a dysfunctional fronto-striatal-cerebellar network in ADHD (Carmona et al., 2005; Kobel et al., 2010; Valera et al., 2007). Additionally, both disorders show a decreased volume and surface area in the corpus callosum. This region is important for the integration of high-level information, for the communication between the two hemispheres and for the sustainment and division of attention (Hutchinson et al., 2008). A structural reduced corpus callosum would therefore compromise long-distance connections and could therefore explain many symptoms related to an aberrant connected brain network.

1.5.3 Previous explanatory models of ADHD and ASD

There are only few attempts to explain ADHD and ASD within one unifying model. One of them is well summarized by Gargaro et al. (2011). They argue that the high comorbidity of ASD and ADHD is greatly explained by the high occurrence of executive dysfunction and abnormalities in regions of the fronto-striatal network. Their explanation is further strongly supported by the fronto-striatal model of Bradshaw (2001). This model describes a disturbance in the fronto-striatal circuitry, which would play a key role in many neurodevelopmental disorders, including ASD and ADHD. As this circuitry comprises the frontal lobe, basal ganglia, thalamus, and cerebellum, it is in high accordance with the findings listed in *Table 1* and *Table 2*.

Another attempt to combine the two disorders in one single model was realized by Sinzig et al. (2009). Their study showed that the consequent phenotypic overlap between ADHD and ASD could be due to different neurochemical pathways. Their Five-Group-Model on the integration of the two disorders enlighten us on the caution needed when studying them together. The five groups consist in a pure ADHD, a pure ASD, an ASD with categorical diagnosis of ADHD, an ADHD with ASD symptoms under the categorical diagnosis threshold, and an ASD group with epiphenomenal ADHD symptoms such as increased stereotyped movements or behaviors.

Both models above support the strong connection and overlap between the two disorders, but fail in providing good explanation for the abnormal brain development found in children with ADHD or ASD. The following section aims at a better explanation of the neurodevelopment course of the two disorders, by accounting for the abnormal structural findings mentioned in *Table 1* and *Table 2*.

1.5.4 My explanatory model of ADHD and ASD

The origin of both neurodevelopmental disorders seems to be linked to an abnormal growth rate during childhood. For both disorders, it is thought that unusual growth trajectories disturb the development of healthy long-distance connections. The absence of the latter leads to a shift for more localized short-range connections. A diminishment of relevant long-distance pathways could account for a dysfunctional fronto-striatal network. In turn, the loss of frontal top-down control on other areas could explain many cognitive disabilities found in both disorders, such as inattention, impulsivity, hyperactivity and executive dysfunction. Because complex connectivity patterns are essential in a normal brain, van den Heuvel & Sporns (2011) investigated regions that show a high degree of interconnection with one another, and a low degree of connection to other regions. On each hemisphere, they found six strongly interconnected "rich club hubs": the precuneus, the superior frontal cortex, the superior parietal cortex, the hippocampus, the putamen and the thalamus. It is believed that such highly interconnected regions help the complex network that is our brain to acquire its small-worldness feature (van den Heuvel & Sporns, 2011). A small-world network is comprised by nodes with many local connections and a few long distance connections, which cause the network to have a high cluster coefficient and a short path length (Watts & Strogatz, 1998). A study conducted on individuals with schizophrenia found decreased connection density among rich club hubs,

suggesting a disruption in global connectivity (van den Heuvel et al., 2013). A similar disruption might be present in children with ADHD or ASD.

But as described, ADHD and ASD are two distinct disorders. They have common characteristics and symptomatology, but seem to differ in their structural abnormalities. The directions of the two growth trajectories clearly diverge, as ASD shows an increased growth rate in early years with a plateauing effect into adulthood (Courchesne, Pierce, Schumann, Redcay, Buckwalter, Kennedy & Morgan, 2007), whereas ADHD shows a decreased growth rate at the beginning, further following a parallel trajectory to healthy subjects into adulthood (Castellanos et al., 2002a).

A good attempt to explain the initial overgrowth found in ASD was done by Markram & Markram (2010). Their intense world theory describes a hyper-plastic and hyper-reactive brain as the cause of ASD. The hyper-plasticity leads to an increased number of connections, which is in turn seen in an increase of volume. These high numbers of locally interconnected neurons, combined with the hyper-reactive property of an autistic brain, lead to the favoring of local connections during activation. Any activation occurring in such an interconnected network has a high probability to strengthen more local connections. This local hyper-activation is like a basin of attraction that keeps the computation local and therefore weakens more long-distance connection. In such enlarged, less organized and less efficiently connected brain, a stimulus with a high arousal level could lead to an intense and painful regional hyper-activation. This might explain why autistic people tend to avoid social stimuli, e.g. faces, as they have notably been reported to be highly arousing (Kleinhans, Johnson, Richards, Mahurin, Greenson, Dawson & Aylward, 2009). Further, the high efficiency of these local connections could explain why ASD children have more problems with planning and shifting attention than children with ADHD, and why they favor restricted and repetitive patterns of behavior. My assumption is that, as this connection overgrowth occurs, necessary associations between more distant regions are eventually established. However, these pathways might not be as straightforward as found in TDC. When connections eventually reach an adequate setup, the overgrowth stops and synaptic pruning takes place. Finally, almost no volume differences can be detected anymore between ASD and TDC people. This hypothesis could explain why people with ASD rarely recover from the disorder, but are capable of learning many unique strategies to compensate for their deficits.

In contrast to that, stands the decreased growth rate found in children with ADHD. The exact cause for this abnormal growth trajectory remains unknown. Analog to the ASD explanation, the initial decreased growth rate in ADHD children could be due to a delayed start or a slower creation of connections throughout the brain. The missed opportunity for establishing necessary connections could lead to a lack of local and more direct long-distant connections. This could account for the local and global decreased brain volume found in ADHD children. My assumption is that, such a brain with general deficit in essential connections would struggle to keep any given activation locally. Thus, this activation would broaden and disperse into other regions. Such mechanism could explain why ADHD children have more problems with inhibition or sustained attention than ASD children, and why people with ADHD are very easily disturbed by external stimulation. It seems that this initial delay of brain growth cannot be fully compensated during childhood. However, as there is no overgrowth of short-range connections, proper long-distance connections will eventually be established and strengthened. This process would lead to the proclaimed parallel growth trajectory during childhood and could explain why the regional and global brain volume is decreased in ADHD. It could also justify the fact that some ADHD loose the diagnostic criteria for the disorder as they become adults.

The cause and progress of the abnormalities in both disorders, described by my model, are very speculative. As there are many important key players that need to occur at the right time in the development of a healthy brain, there are also high risks of improper development, such as disturbed neurogenesis, errors in cell migration, abnormal cell death, abnormal production of non-neuronal brain tissues, abnormal synaptic pruning, decreased dendritic branching or abnormalities in myelination (Bauman & Kemper, 2005; Penn, 2006). In addition, increased or decreased brain volume can be explained by many other factors than the amount of connections. The variation in volume could be explained by the number and size of neurons, glial cells, afferent and efferent fibers or by the change in density of vasculature. In turn, all these factors are influenced by genetics, growth factors and hormones (McAllister, 2000).

The development of the human brain is very complex. Thus, this study cannot explain the exact neurological cause for ADHD or ASD. However, it can analyze the appearance of the structural abnormalities and provide some insights on how these two disorders are affected in their neurodevelopmental course.

1.6 Hypotheses

Many of the previous findings were acquired through VBM studies, and therefore were restricted to the analysis of volumetric differences in the human brain. However, the human cerebral cortex is a "2-dimensional sheet" highly folded in a 3-dimensional space (Fischl, Sereno, Dale, 1999a). Thus, its main feature is not defined by volume, but rather by thickness and area. As volume is the product of thickness by area, volumetric differences can be caused by either one of the latter. Both of them represent more accurately the actual cytoarchitecture present in the cortex, and give us information about its integrity (Makris et al., 2007). A variation in each of the cortex's six layers could explain changes in cortical thickness (Brodmann, 1909). In a normal human brain, cortical thickness varies between 1 and 4.5 mm with an overall average of about 2.5 mm, and regional variations can be quite large (Fischl & Dale, 2000). Cortical area is defined by the number, the size and the density of its minicolumns, i.e. vertical clusters of highly interconnected cells that extend through the cortical layers. They are thought to be the functional sub-units of the cortex (Stanfield et al., 2008). Abnormalities of minicolumn properties have already been shown in individuals with autism. Indeed, they were found to have an atypical number and width of minicolumns, as well as an increased neuronal density, particularly in the prefrontal cortex (Hyde et al., 2010).

VBM studies cannot grasp the complex topology and geometry of the human cortex, and are therefore restricted to information about size and position. A sensitivity test on VBM studies on ADHD subjects showed that less than half of the studies agreed on the results, indicating a high variability between VBM studies (Frodl & Skokauskas, 2012). As alternative, the surface-based morphometry (SBM) approach centers much more on the actual topographic appearance of the cortex and is capable of providing additional information (Jiao et al., 2010). By comparing the capability of classifying individuals to either ASD or controls, Jiao et al. (2010) found that thickness-based classifications outperformed volume-based classification for each combination of classifier and performance metric.

The goal of the present study is to analyze the structural abnormalities found in children with ADHD or ASD by using an SBM approach. Many studies investigated volume differences in these disorders, but only little has been done on analyzing cortical thickness and cortical surface area. To my knowledge, only one VBM study (Brieber et al., 2007) and no SBM study directly compared structural differences between ADHD and ASD. Therefore, this study is the first one to directly compare children with ADHD to children with ASD, consid-

ering cortical thickness and surface area. With 700 subjects (N_{ADHD} = 173, N_{TDC_ADHD} = 271, N_{ASD} = 115, N_{TDC_ASD} = 141), this study is also the biggest one to investigate volume differences between ADHD and ASD and to look at cortical thickness and surface area differences in either of the two disorders. This new approach has the potential to provide deeper insight in these disorders and obtain further knowledge on the comorbidity standing between ADHD and ASD.

By analyzing the structural MRI (sMRI) scans of children with ADHD, with ASD or TDC, this study tries to answer the following four main questions:

1. What are the structural brain differences between children with ADHD and TDC_{ADHD}?
2. What are the structural brain differences between children with ASD and TDC_{ASD}?
3. What are the structural brain differences between children with ADHD and children with ASD?
4. What are the structural brain similarities between children with ADHD and children with ASD?

Comparisons will be done on the measurements of cortical, subcortical and cerebellar volume, cortical thickness and cortical surface area.

In accordance with previous literature (*Table 1* and *Table 2*), the following findings are expected:

- Compared to TDC_{ADHD}, children with ADHD should have a decreased total cortical volume, mean cortical thickness and total cortical surface area. These decreases should be found throughout childhood and adolescence.
- Compared to TDC_{ADHD}, children with ADHD should show regional abnormalities throughout the brain, as described by *Table 1* and *Table 2*.
- Compared to TDC_{ASD}, children with ASD should have an initially increased total cortical volume and mean cortical thickness. As the increased growth rate is mostly restricted to early years, and as a plateauing and decreasing growth rate is expected with age, global differences are expected to be present only in children (8 to 12 years) and to be absent in adolescence (13 to 18 years).
- Compared to TDC_{ASD}, Children with ASD should show regional abnormalities throughout the brain, as described by *Table 1* and *Table 2*.

- In accordance with the aberrant connectivity theory, both disorders could show a decreased volume of the corpus callosum. This effect is supposed to be more pronounced in anterior regions, as this part projects into frontal cortical areas, which are associated with the executive dysfunction theory in both disorders.

To solve the incoherence found in previous literature, the study at hand will also try to answer the following questions: Is abnormal brain size in ADHD or ASD mainly driven by gray or white matter, or are they both equally involved? This question is raised, as opinions about the driving force behind the enlarged autistic brain are very diverging (Bonilha, Cendes, Rorden, Eckert, Dalgalarrondo, Li & Steiner, 2008; Courchesne et al., 2007, Herbert et al., 2004). This question will be assessed by looking separately at the total volume of gray and white matter and see if the ratio between them is abnormal in children with ADHD or ASD, compared to their respective TDC.

To account for the strong influence of confounding factors, the following parameters will be used as covariates. It must be stressed that this control is relevant, as the groups used in this study were not matched on those factors, which could lead to strong confounding influence on the results:

- *Age*: Age is probably the most influential factor in the study of structural brain abnormalities in children with a neurodevelopmental disorder.
- *IQ*: There seems to be a strong relationship between IQ and brain volume, cortical thickness or cortical surface area. Studies in healthy individuals reported that a thinner cortex, a larger surface area or an increased gray matter volume correlate with a higher IQ, and that age-dependent structural changes are stronger in people with higher IQ (Haier, Jung, Yeo, Head & Alkire, 2004; Schnack et al., 2014). To account for those influences, IQ has to be used as a covariate.
- *Gender*: Most previous findings were solely conducted on male populations. The population in this study also includes females, and therefore has to account for it. The importance of controlling for gender is supported by a meta-analysis reporting regional deviating findings if gender was either accounted for or not (Frodl & Skokauskas, 2012).
- *Total gray matter volume, total cortical surface area* and *mean cortical thickness*: It is unclear whether abnormalities in ADHD or ASD are based on regional or global changes. There are substantial findings mentioned in *Table 1* that were not significant any-

more once total brain volume was accounted for. To assess the influence of a global change and to account for it, total gray matter volume, total cortical surface area or mean cortical thickness were used as covariates. For the analysis of each cortical measurement, the corresponding global measurement covariate was used. It must be stressed here that total gray matter volume consists of subcortical and cerebellar gray matter volume, and that total cortical surface area is measured on the cortical white matter surface.

2. Methods

2.1 Dataset

All data for this study was gathered from two databases, the ADHD-200 database (http://fcon_1000.projects.nitrc.org/indi/adhd200/) and the Autism Brain Imaging Data Exchange (ABIDE) database (http:// http://fcon_1000.projects.nitrc.org/indi/abide/). The data is publicly available and belongs to a bigger data consortium called 1000 Functional Connectomes Project (http://fcon_1000.projects.nitrc.org). Both databases provided, in almost all cases, at least one structural and one rsfMRI scan per subject, in addition to information about the age at scanning time, IQ and gender. The subjects from both databases stemmed from multiple scanner sites and had already been partly used in other studies. More information about these studies can be found on the ADHD-200 and ABIDE homepage. However, no study analyzing the whole ABIDE dataset had been conducted so far, and the studies related to ABIDE almost exclusively focused on the rsfMRI data. The ADHD-200 dataset was used in a similar way, but some studies that analyzed the whole dataset already exist (e.g. Eloyan et al., 2012; Olivetti, Greiner & Avesani, 2012). Most of them came from the "ADHD-200 Global Competition", held in September 2011. The goal of this competition was to establish a classifier that could assign a subject either to an ADHD or to a control group, given the data provided by the ADHD-200 dataset. These studies used machine-learning algorithms to artificially generate a classifier able to distinguish children with ADHD from TDC. It must be stressed that the goal of these studies was to maximize the classifier accuracy with the intention of winning the competition (Eloyan et al., 2012). Moreover, these studies that analyzed the sMRI scans used a VBM approach and did not use cortical surface information, such as thickness or surface area. Thus, as no cortical surface information was used and as these studies did not use an empirical-based approach to look at difference between children with ADHD and TDC, I see no possible conflict in using the whole ADHD-200 dataset for the report at hand. To my knowledge, this is the first study analyzing the ADHD-200 and the ABIDE dataset together.

The data for this study was downloaded on the 1st of November 2013. At that time, the ADHD-200 dataset consisted in 973 subjects from 8 different scanner sites. The ABIDE dataset contained 1112 subjects of 17 different scanner sites. For more detailed information about the composition of the two databases, see *Supplementary A1*. It must be stressed that this total dataset of 2085 subjects also included people that were irrelevant to the present study (e.g. subjects older than 18 years), scanner sites with a lack of important information or

scans of doubtful quality. Therefore each subject, scanner site and sMRI scan had to reach certain criteria to be included in the final dataset. The following sections describe these criteria.

2.1.1 Subject criteria

Subjects had to fulfill the following five criteria to be included in the final dataset. *First*, as this study focuses on children, subjects had to be aged of 8.0 to 18.0 years. This criterion led to the exclusion of 476 subjects. *Second*, the main diagnosis of a subject had to be known. That is, a subject had to be labeled either as ADHD, ASD or TDC. A list of the diagnosis tool used by each scanner site included in the final dataset can be found in *Supplementary A2*. This criterion led to the exclusion of another 26 subjects. *Third*, the gender and IQ of a subject had to be known, as those factors were used as covariates. This criterion led to the exclusion of 53 additional subjects. The IQ measurement used in this study was the full scale IQ (FSIQ) (Wechsler, 2003). For a list of the IQ measurement used by the scanner sites included in the final dataset, see *Supplementary A3*. For 37 subjects, the FSIQ had to be calculated from the performance and verbal IQ according to the manual of the given IQ measurement. *Fourth*, as all sites used a different IQ threshold to exclude subjects with too low score from their dataset, the IQ threshold of this study was set to 85, as this was the highest of the lower threshold of all scanner sites, used by the University of Michigan. This criterion led to the exclusion of another 94 subjects. *Fifth*, subjects had to be free of any comorbidity that could have led to its affiliation with another diagnostic group. That is, a subject with ASD was excluded if also having ADHD and vice versa, or if a TDC was borderline ADHD. This criterion resulted in the exclusion of 19 subjects.

The implementation of the subject criteria resulted in the exclusion of 668 subjects and reduced the population from 2’085 to 1417 individuals.

2.1.2 Scanner site criteria

Although all sites’ scanners used a magnetic field of 3 Tesla, there is still a fair amount of confounding heterogeneity expected between different scanner sites, due to different scan parameters and scanner specific differences. More information about the specific scanner parameters per site of the subjects included in the final dataset can be found in *Supplementary A4*. To control for the influence of scanner specific characteristics, a site had to provide at

least with 5 subjects in either of the groups ADHD, ASD or TDC. If not, the whole site was excluded from the analysis. Therefore, the following scanner sites were excluded: California Institute of Technology (N = 2; only 1 subject with ADHD left), NeuroIMAGE sample (N = 6; only 4 subjects with ADHD left), Washington University in St. Louis (N = 46; only TDC), and the ADHD set of University of Pittsburgh (N = 75; only 4 subjects with ADHD left).

The implementation of the scanner site criteria led to the exclusion of 129 subjects and reduced the population from 1'417 to 1'288 individuals.

2.1.3 Dataset criteria

As this study is based on anatomical MRI scans, subjects provided only with functional resting state MRI scans were excluded from the final dataset. This resulted in the exclusion of 6 subjects (4 with Autism and 2 TDC).

The ABIDE homepage (http://fcon_1000.projects.nitrc.org/indi/abide/) mentions that some of the subjects in the TDC group were also uploaded as control subjects to the ADHD-200 database. Therefore a thorough control for doubles was conducted. Thus, 42 subjects were found to be included twice in this study. Of these 42 doubles, 41 were from the scanner site of New York. They were all uploaded as TDC once for the ADHD-200 set of New York University Child Study Center and once for the ABIDE set of NYU Langone Medical Center. To keep the final ratio between subjects with disorder and TDC, per site, close to a 1:1 ratio, almost all the 41 doubles were accounted to the ADHD-200 dataset. An exception was made when the following preprocessing criteria excluded a double from the ADHD-200 dataset, but not from the ABIDE dataset. Thus, 6 doubles were accounted to the ABIDE dataset. This distribution of TDC resulted in an ADHD to TDC ratio of 113 to 82 in the New Yorker ADHD-200 dataset, and in an ASD to TDC ratio of 41 to 32 in the New Yorker ABIDE dataset.

Two additional subjects from the University of Michigan dataset were uploaded with the identical sMRI scan, but different demographic characteristics. Both subjects were therefore excluded from the final dataset.

The implementation of the dataset criteria resulted in the exclusion of 49 subjects and reduced the population from 1'288 to 1'239 individuals.

2.1.4 Preprocessing criteria

To get subject's specific information on brain volume, cortical thickness and surface area, the data first had to be preprocessed. A detailed description on how the preprocessing was conducted can be found in the next section. However, if the sMRI scan of a subject could not be preprocessed or failed the given quality checks, subject was excluded from the final dataset. There were three preprocessing criteria: *First*, a subject was excluded from the dataset if the reconstruction algorithm (i.e. recon-all) of the FreeSurfer software suite (implemented by Fischl and Dale (2000); https://surfer.nmr.mgh.harvard.edu/fswiki) resulted in any error that could not be handled without a manual user intervention. This resulted in the exclusion of 11 subjects. *Second*, the anatomical scan of a subject had to have a signal-to-noise ratio (SNR) of at least 16 to ensure a certain level of data quality. The SNR was calculated by FreeSurfer's software package called QA Tools (https://surfer.nmr.mgh.harvard.edu/fswiki/QATools), where the threshold of 16 is used as default parameter. A low SNR can be found because of magnetic field inhomogeneity or movement in the scanner (Yeo et al., 2011). This resulted in the exclusion of 367 subjects. *Third*, if the cortical parcellation or the subcortical segmentation done by FreeSurfer's recon-all algorithm was not sufficient,, the subject was excluded. Two insufficient and two sufficient examples can be found in *Supplementary A5*. The subcortical segmentation and cortical parcellation algorithms failed if they could not distinguish the brain tissue from the surrounding cerebrospinal fluid or the skull. This can occur because of the rapid magnetic susceptibility changes between different types of tissues (gray matter, cerebrospinal fluid, and scull) and was most often seen to happen in basal regions of the temporal lobe. The outcome of the parcellation and segmentation procedure of each subject was rated by two independent raters and subjects were excluded if at least one rater declared either the segmentation or the parcellation as insufficient. This resulted in the exclusion of 116 subjects.

The implementation of the preprocessing criteria led to the exclusion of 494 subjects and reduced the population from 1'239 to 745 subjects.

In this dataset of 745, only six subjects with ADHD-H, stemming from four different scanner sites remained. Because of this low number of individuals affected by this disorder's subtype, all six were excluded from the final dataset. Further, as the ASD subtype PPD-NOS was not relevant for this study, the 14 subjects with this subtype, stemming from five different scanner sites, were also excluded from the final dataset.

After applying all those exclusion criteria, some scanner sites contained less than 5 subjects in one of the groups ADHD, ASD or TDC. Therefore the scanner site criterion of having at least 5 subjects in either group was applied again. This led to the exclusion of the following scanner sites: Ludwig Maximilians, University Munich (N = 7, only 4 subjects with ASD left), Yale School of Medicine, Child Study Center (N = 4, only 1 subject with ASD left), Olin, Institute of Living at Hartford Hospital (N = 10, only 4 subject with ASD left), San Diego State University (N = 4, only TDC left).

2.1.5 Final dataset

The application of all exclusion criteria to the initial dataset resulted in a final dataset of 700 subjects. For an overview of the exact numbers of exclusions per criteria from the previous sections see *Supplementary A6.* The following *Table 3* shows the composition of the final dataset and summarizes the characteristics of the four groups ADHD, ASD, healthy controls for ADHD (TDC_{ADHD}) and healthy controls for ASD (TDC_{ASD}). For a more detailed version of the characteristics, broken down for each site, see *Supplementary A7.* The two healthy control groups were kept as two separate groups because their scans were acquired from different scanner sites. Therefore, it cannot be assumed that the two populations of TDC come from the same population.

Table 3 *Characteristics of the groups ADHD, TDC_{ADHD}, ASD and TDC_{ASD} in the final dataset*

	Subjects [#]	*Gender [m/f]*	*Age [year]*	*IQ [FSIQ]*
ADHD-200	444	272 / 172	11.08 (2.22)	112.56 (12.89)
TDC_{ADHD}	271	138 / 133	11.00 (2.10)	115.34 (12.36)
ADHD in total	173	134 / 39	11.22 (2.39)	108.22 (12.54)
ADHD-C	96	79 / 17	10.65 (2.13)	109.23 (12.79)
ADHD-I	77	55 / 22	11.93 (2.51)	106.96 (12.17)
ABIDE	256	225 / 31	12.95 (2.67)	109.67 (12.24)
TDC_{ASD}	141	121 / 20	12.78 (2.74)	111.76 (11.46)
ASD in total	115	104 / 11	13.17 (2.59)	107.10 (12.72)
Autism	94	84 / 10	13.41 (2.61)	105.43 (11.54)
Asperger's	13	12 / 1	12.67 (2.21)	113.00 (16.93)
Subtype not known	8	8 / 0	11.14 (2.11)	117.16 (12.64)
Total dataset	700	497 / 203	11.77 (2.56)	111.50 (12.73)

The column age and IQ represent the mean of the sample with the standard deviation written inside brackets.

2.2 Analysis of anatomical scans

2.2.1 Preprocessing

The preprocessing of the sMRI data was done in four steps. *First*, a cortical surface reconstruction and volumetric segmentation of each subject's sMRI scan was conducted, using FreeSurfer image analysis suite, version 5.3.0. FreeSurfer is a freely available software package, downloadable at http://surfer.nmr.mgh.harvard.edu/ and is mainly used for cortical surface-based analysis. The technical details of these procedures are described in prior publications (Dale, Fischl, Sereno, 1999; Fischl et al. 1999a; Fischl, Sereno, Tootell, Dale, 1999b; Fischl & Dale, 2000; Fischl, Liu, Dale, 2001; Fischl et al., 2002; Fischl et al., 2004a; Fischl et al., 2004b; Ségonne, Dale, Busa, Glessner, Salat, Hahn, Fischl, 2004). Briefly, FreeSurfer takes the sMRI scan of a subject and reconstructs a subject-specific cortical surface model that contains information such as cortical thickness, cortical surface area and cortical volume. The fully automated software pipeline involves motion correction (Reuter, Rosas, Fischl, 2010), removal of non-brain tissue, (Ségonne et al., 2004), segmentation of subcortical white matter and deep gray matter structures (Fischl et al., 2002, 2004a), intensity normalization (Sled, Zijdenbos, Evans, 1998), tessellation of the gray matter white matter boundary and automated topology correction (Fischl et al., 2001; Ségonne, Pacheco, Fischl, 2007). Then, a surface deformation algorithm finds the borders between gray matter, white matter and cerebrospinal fluid, by calculating the intensity gradient shift that takes place between different tissue classes (Dale et al., 1999; Dale and Sereno, 1993; Fischl & Dale, 2000). The borders between the tissues are not restricted to the fixed voxel grid of a sMRI scan, and are therefore capable of detecting submillimeter differences. By using intensity and continuity information from the surface deformation procedure, FreeSurfer is capable to interpolate surface locations for regions for which the MRI scan is ambiguous (Fischl & Dale, 2000). Procedures for the measurement of cortical thickness have been validated with histological analysis (Rosas et al., 2002) and manual measurements (Kuperberg et al., 2003; Salat et al., 2004). FreeSurfer morphometric procedures have been demonstrated to show good test-retest reliability across scanner manufacturers and across field strengths (Han et al., 2006; Reuter, Schmansky, Rosas, Fischl, 2012). The recreated model of the surface further gets inflated (Fischl et al., 1999a) and a parcellation of the cerebral cortex, dividing the surface into units based on gyral and sulcal structure, takes place (Desikan et al., 2006; Fischl et al., 2004b). From the parcellated surface model, variety of surface based data can be extracted, such as cortical surface area, cortical thickness and cortical volume. Cortical thickness is defined as the closest distance from the gray/white boundary to the gray/CSF boundary at each vertex on the tessellated sur-

face (Fischl & Dale, 2000). As the thickness maps use spatial intensity gradients across the tissue classes, they are not reliant on absolute signal intensity and are sensitive enough to detect submillimeter differences (Fischl & Dale, 2000).

The preprocessing of the sMRI data of this study was achieved without any manual user intervention, as the FreeSurfer software suite is fully automated. On an average multi-core desktop PC, the completion of the recon-all algorithm for the 700 subjects would have taken at least two months, as the computation time per subject is of about 8 hours. Luckily, I was able to execute this computation on a supercomputer at the University of Zurich (http://www.uzh.ch) called Schrödinger HPC (http://www.scicomp.uzh.ch/schroedinger.html). This cluster offers 4608 processor cores (Intel Xeon 5500; 2.8 GHz) on 576 compute nodes, each having 24GB RAM, that are connected over QDR Infiniband (40 Gbit/s) and attached to a luster file system with 300 TB disk space. With such a powerful machine, the computation was completed within three days.

After completion of this computation, the following measurements were available for each subject: measurements of cortical volume, thickness and surface area throughout the brain, as well as of 74 regions per hemisphere, according to the atlas by Destrieux, Fischl, Dale and Halgren (2010), subcortical and overall volume according to the atlas by Fischl et al. (2002). As previous literature only investigated lobular differences, corresponding ROIs had to be created. Using the parcellation atlas by Desikan et al. (2006), ROIs for the frontal, temporal, parietal, occipital, cingulate and insular lobe were created. Further, measurements of cortical thickness, surface area and volume of each lobular ROI were obtained.

Second, a surface, curvature and volume average was created out of the 700 subjects of the final dataset. *Third*, each subject was resampled to this average brain, in order to have the data present in a common reference space. *Fourth*, the data was smoothed on the surface with a smoothing kernel of full width at half maximum (FWHM) of 10mm. The smoothing kernel was set to this value as it can be assumed that the high number of subjects already provides some degree of smoothing effect on the resulting group analysis.

2.2.2 Statistical Modeling

A general linear model (GLM) approach was used to analyze the global and regional differences and similarities in the cortical and subcortical volume, cortical thickness and cortical surface area between the four groups ADHD, TDC_{ADHD}, ASD, and TDC_{ASD}. Age, gender, total gray matter volume and IQ were found to make a significant contribution to the model and were thus included as covariates. The covariates are assumed to have different on-

sets, but same slopes throughout all groups. The data was analyzed on the whole surface using a vertex-wise analysis. The estimated contrasts to compare the groups are as follows:

1. An F-contrast to investigate differences between any two groups
2. A T-contrast to investigate differences between ADHD and TDC_{ADHD}
3. A T-contrast to investigate differences between ASD and TDC_{ASD}
4. A T-contrast to investigate differences between ASD and ADHD
5. A T-contrast to investigate differences between children with either disorder and TDC. This contrast was created by combining ASD and ADHD into one group and comparing them to a group containing all TDCs.

2.2.3 Statistical inference

The statistical inference in the whole brain vertex-wise analysis was done with the tools provided by FreeSurfer's software suite (https://surfer.nmr.mgh.harvard.edu/fswiki). Two-sample t-tests with pooled variance estimates were used, and each vertex was thresholded at $p < 0.05$. To control for the issue of multiple comparisons, a non-parametric cluster size correction was performed by using 10'000 synthetic z-map permutations (Monte Carlo simulations) with a vertex-wise cluster-forming threshold of $p < 0.05$.

All ROI-wise comparisons, as well as the comparisons of age, IQ and gender, were performed by means of SPSS 22.0 (http://www.spss.com) for Linux. The following three comparisons were conducted: ADHD versus TDC_{ADHD}, ASD versus TDC_{ASD} and ADHD versus ASD. Age and IQ were compared by using an independent t-test, gender by using a Pearson's chi-squared test and the ROIs by using an analysis of covariance (ANCOVA). As previously mentioned, age, IQ, gender and either total gray matter volume (TGMV), total cortical surface area (TCSA) or mean cortical thickness (MCT) were used as covariates in the ANCOVA. TGMV was used as covariate in all models of volume measurements, TCSA in all models concerning surface area measurements and MCT in all models of mean thickness measurements.

3. Results

As shown by the results in *Table 1*, there is a significant age difference between the group of children with ADHD and the group of children with ASD. Because of this finding, and because of the assumption that both disorders manifest themselves in younger age differently than in older years, the four groups were split by the mean age of 13 years, creating the additional subgroups *Age 8 to 12* and *Age 13 to 18*. Each analysis was performed on the main group, as well as on the two subgroups. *Figure 1* summarizes the number of subjects per group and subgroup.

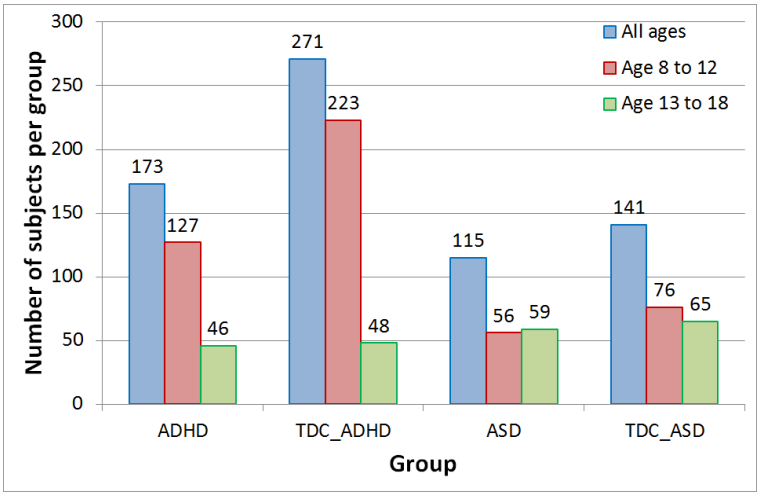

Figure 1. *Number of subjects per group and subgroup.*

3.1 Analysis of age, IQ and gender

As shown in *Figure 2* and *Supplementary B1*, the group of children with ADHD is significantly younger than the group of children with ASD, $t_{(df=286)}$ = -6.54, p = 2.8E-10. This difference remains significant, even after splitting the groups at the age of 13 years. However, both disorder groups did not differ significantly from their controls. With respect to IQ, both ADHD and ASD showed a significantly lower IQ than their controls, but did not differ significantly between one another ($t_{(df=286)}$ = 0.74, p = 0.46). The difference in IQ between ASD and TDC_{ASD} is no longer significant if considering only the older subgroup of adolescence ranking between 13 and 18 years old. Finally, there was a significantly ($\chi^2_{(1,\ N\ =\ 444)}$ = 33.33, p = 2.2E-8) higher number of girls in the TDC_{ADHD} group (49%) compared to the ADHD group (23%), and a significantly ($\chi^2_{(1,\ N\ =\ 288)}$ = 8.11, p = 0.004) higher number of girls in the ADHD (23%) group compared to the ASD group (10%).

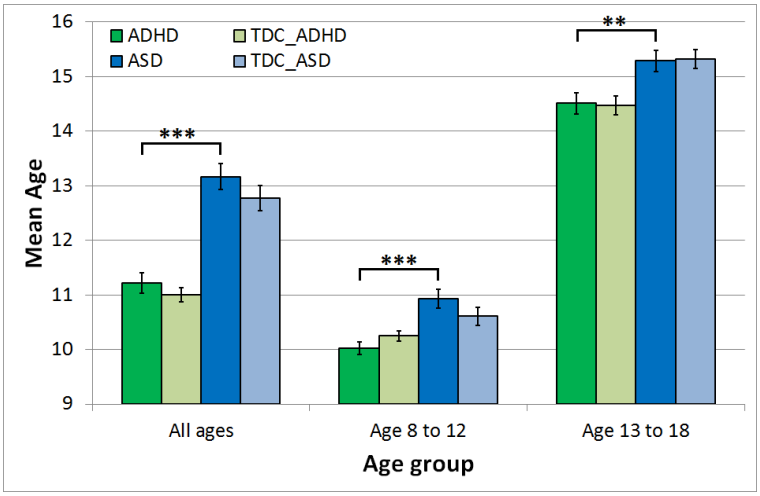

Figure 2a. *Mean age of all groups and subgroups. Error bars show mean standard error. Significant differences between groups are encoded as follows: ** = p < 0.01; *** = p < 0.001. For more information see Supplementary B1.*

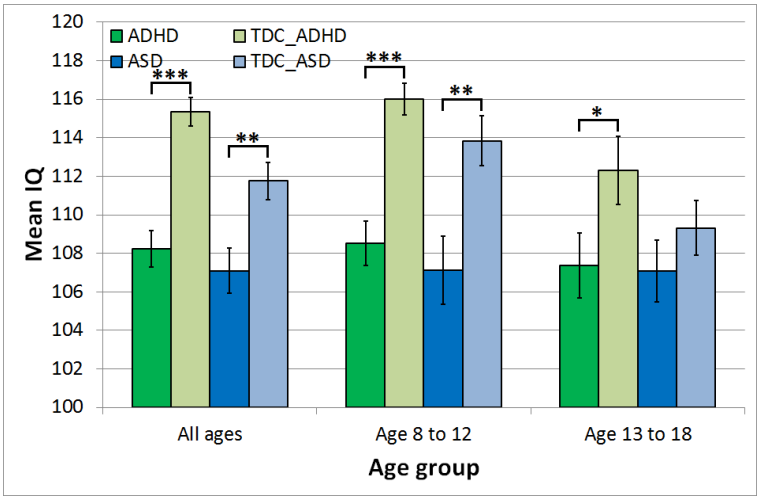

Figure 2b. *Mean IQ of all groups and subgroups. Error bars show mean standard error. Significant differences between groups are encoded as follows: * = p < 0.05; ** = p < 0.01; *** = p < 0.001. For more information see Supplementary B1.*

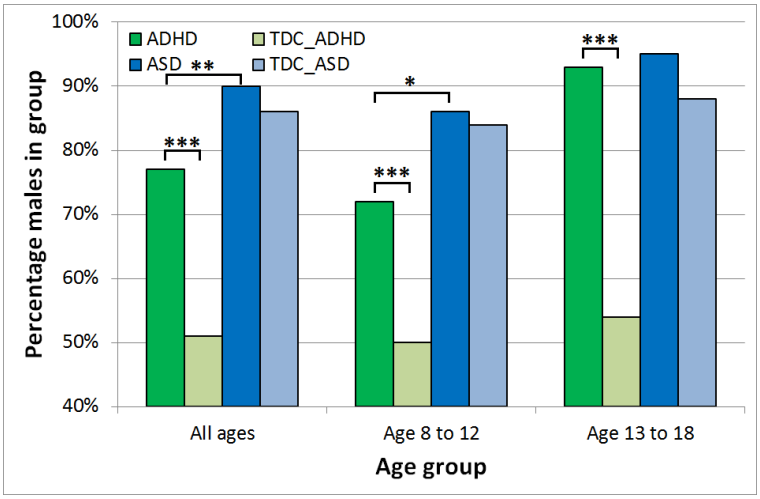

Figure 2c. *Percentage males in all groups and subgroups. Significant differences between groups are encoded as follows: * = p < 0.05; ** = p < 0.01; *** = p < 0.001. For more information see Supplementary B1.*

3.2 Analysis of global brain measurements

The analysis of global cortical GM and WM measurements is shown in *Figure 3* and *Supplementary B2*. Gender, age and IQ were used as covariates in all estimations. Children with ADHD had a significantly smaller TGMV than TDC_{ADHD}, $p = 0.044$, and than ASD, $p = 0.002$. This difference seemed to be mostly driven by an effect in younger years, as the significant difference could also be seen in children aged of 8 to 12 years. The comparison of total white matter volume (TWMV) showed no significant difference between groups. The comparison of mean cortical thickness showed that children with ADHD had a significantly thinner cortex than children with ASD, $p = 0.003$ and that this effect was mainly driven by a change in adolescence. The comparison of total cortical surface area (TCSA) revealed a significantly reduced surface area between ADHD and TDC_{ADHD}, $p = 0.007$, but only in the younger group, as this difference was not present in the older group, $p = 0.84$. No significant difference between ADHD and TDC_{ADHD}, ASD and TDC_{ASD} and ADHD and ASD was found in cerebellar gray or white matter volume (see *Supplementary B3*).

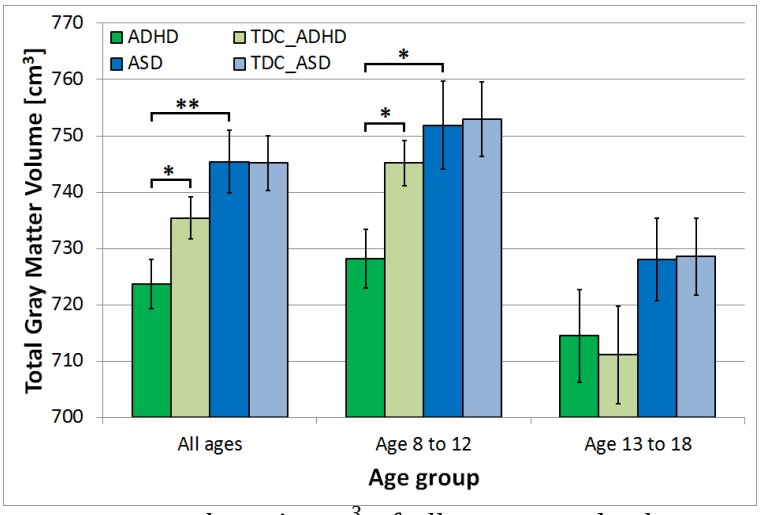

Figure 3a. *Total gray matter volume in cm^3 of all groups and subgroups. Error bars show mean standard error. Significant differences between groups are encoded as follows: * = $p < 0.05$; ** = $p < 0.01$. For more information see Supplementary B2.*

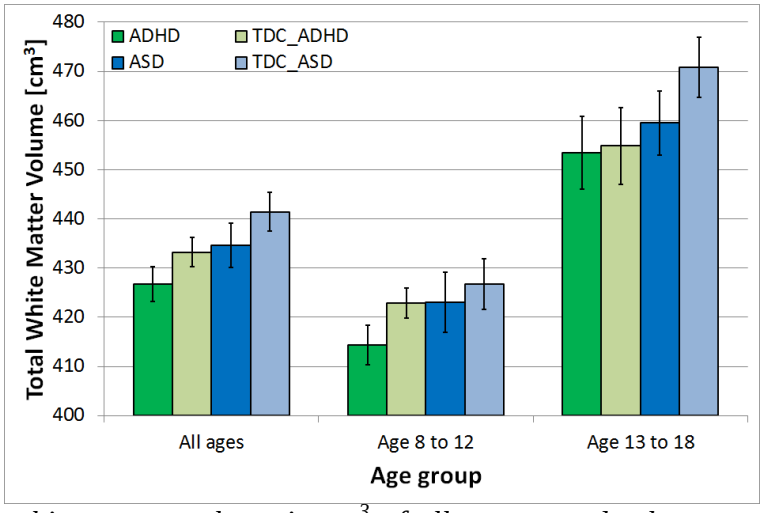

Figure 3b. *Total white matter volume in cm^3 of all groups and subgroups. Error bars show mean standard error. For more information see Supplementary B2.*

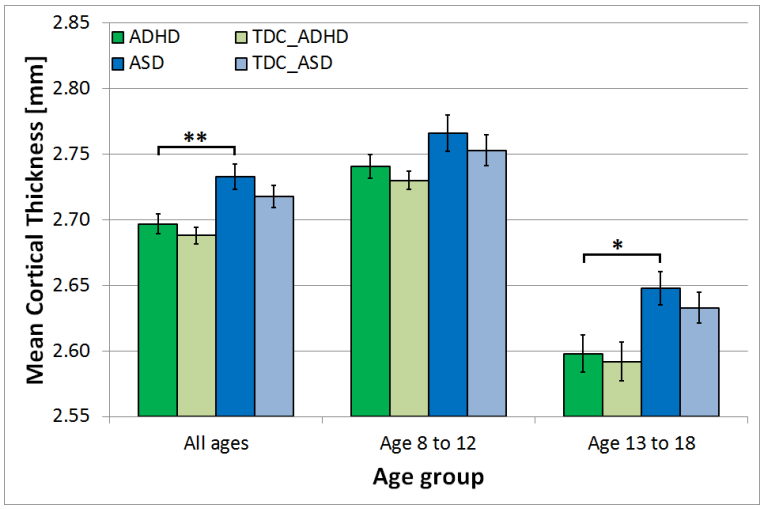

Figure 3c. *Mean cortical thickness in mm of all groups and subgroups. Error bars show mean standard error. Significant differences between groups are encoded as follows: * = p < 0.05; ** = p < 0.01. For more information see Supplementary B2.*

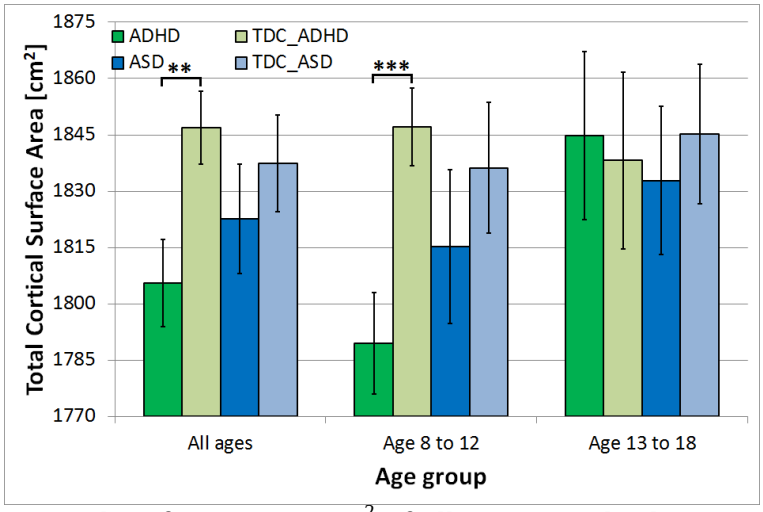

Figure 3d. *Total cortical surface area in cm^2 of all groups and subgroups. Error bars show mean standard error. Significant differences between groups are encoded as follows: ** = p < 0.01; *** = p < 0.001. For more information see Supplementary B2.*

An analysis of the cortical and subcortical GM to cortical WM volume ratio between the groups only showed a trend of increased cortical and subcortical GM volume, compared to cortical WM volume in ASD compared to TDC_{ASD}, $p = 0.09$, see *Supplementary B4*. No other significant difference was found. This trend of increased GM volume compared to cortical WM volume seems to fade away by the time of adolescence.

3.3 Analysis of lobe measurements

The analysis of lobar measurements revealed multiple findings, described below. Gender, age and IQ were used as covariates in all estimations. Additionally, TGMV was used as covariate in the estimation of cortical volume measurements, TCSA as covariate in the estimation of cortical surface area, and mean cortical thickness as covariate in the estimation of cortical thickness measurements. For a detailed view on the cortical thickness, cortical surface area and cortical volume of each lobe, refer to *Supplementary B5a-f*.

FRONTAL LOBE

The analysis of the **frontal lobe** showed a significantly increased cortical thickness in ADHD compared to TDC_{ADHD} ($p = 0.022$), and that this increase was strongly driven by differences in younger years ($p = 0.005$). Children with ADHD had a significantly increased cortical thickness in comparison to children with ASD ($p = 1.6E\text{-}6$). This increased cortical thickness could be observed in younger ($p = 0.007$), as well as in older children ($p = 0.001$). Children with ADHD additionally showed a significant decrease in cortical surface area compared to ASD ($p = 0.007$), but this effect seemed to be mostly based on differences in younger years ($p = 0.045$).

TEMPORAL LOBE

The analysis of the **temporal lobe** showed that children with ADHD had, in contrast to $TDC_{ADHD,}$ a significantly decreased cortical thickness ($p = 1.2E\text{-}4$) and cortical volume ($p = 0.004$), and that this difference was mostly driven in differences in younger years ($p = 1.5E\text{-}4$ for thickness and $p = 4.0E\text{-}4$ for volume). Additionally, a decreased cortical surface area ($p = 0.041$) could be observed in younger children with ADHD in contrast to their controls. Children with ASD, in contrast to $TDC_{ASD,}$ showed a significant decrease only in cortical thickness ($p = 0.049$).

PARIETAL LOBE

The analysis of the **parietal lobe** showed significant increase in cortical surface area in ADHD compared to TDC_{ADHD} (p = 0.018) and compared to ASD (p = 0.014). The difference between ADHD and TDC_{ADHD} seems to be driven in a difference in younger years (p = 0.041) while the difference between ADHD and ASD seems to be most prominent in older childhood (p = 0.001). Additionally, a significant increase in cortical volume could be observed in children with ADHD, in contrast to ASD (p = 0.034), but only in older years.

OCCIPITAL LOBE

The analysis of the **occipital lobe** showed significantly increased cortical thickness in ADHD compared to TDC_{ADHD}, (p = 0.031). Children with ADHD showed, in contrast to ASD, a significantly decreased cortical thickness (p = 0.002). This difference between the two disorders was mostly driven by differences in later years (p = 0.040), but this trend could also be seen in younger children (p = 0.066). Children with ASD showed, in contrast to $TDC_{ASD,}$ an increased cortical surface area (p = 0.021) and a significantly decreased cortical area in contrast to ADHD (p = 0.048), but only in younger years.

CINGULATE CORTEX

The analysis of the **cingulate cortex** showed a significantly decreased cortical thickness in ADHD compared to TDC_{ADHD} (p = 0.003) and compared to ASD (p = 1.2E-5). The difference to TDC_{ADHD} was mostly based on differences in younger years (p = 0.007), while the differences to ASD was driven by differences in later years (p = 5.0E-6). The analysis on the cortical volume showed that children with ADHD had, in contrast to $TDC_{ADHD,}$ a decreased cortical volume (p = 0.002) and that this decrease was mainly driven by differences in younger years (p = 0.019). A significant decreased volume in ADHD, in contrast to ASD, could only be seen in older children (p = 0.023). The analysis also revealed that younger children with ASD have, in contrast to younger children with ADHD, a significantly decreased cortical surface area (p = 0.047).

INSULAR CORTEX

The analysis of the **insular cortex** showed a significantly decreased cortical thickness in ADHD compared to TDC_{ADHD} (p = 4.6E-4) and compared to ASD (p = 0.006). Both differences were strongly driven by changes in older years (p = 0.016 for the contrast to TDC_{ADHD} and p = 0.002 for the contrast to ASD). Additionally, the significant decreased thickness in

ADHD, compared to TDC_{ADHD}, could also be observed in younger children ($p = 0.008$). Children with ADHD also showed a significant decreased in insular volume compared to TDC_{ADHD} ($p = 0.004$), which was mostly driven by differences in younger years ($p = 0.015$).

3.4 Analysis of corpus callosum measurements

Since the corpus callosum is a brain structure consisting of white matter fibers, total cortical white matter volume was used as covariate, beside the usual covariates: gender, age and IQ. The five regions anterior, central, mid-anterior, mid-posterior and posterior were analyzed separately. Additionally, by summing up the volume of all regions into a total corpus callosum volume, the whole structure was also analyzed as one. For a detailed view on the corpus callosum volume, see *Supplementary B6*.

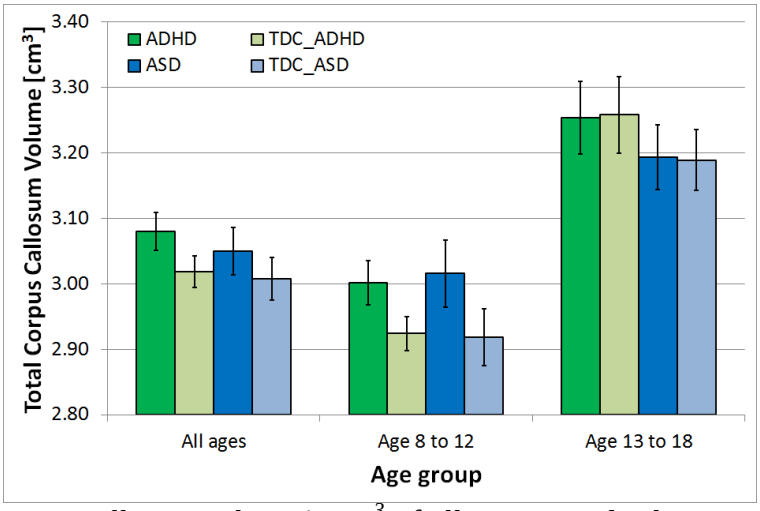

Figure 4. *Total corpus callosum volume in cm^3 of all groups and subgroups. Error bars show mean standard error. For more information see Supplementary B6.*

The analysis of the total corpus callosum volume did not show any significant difference between any groups (*Figure 4*). But there was a trend towards a decreased total corpus callosum volume in younger children with ADHD ($p = 0.07$), in contrast to $TDC_{ADHD,}$ or in younger children with ASD ($p = 0.14$), in contrast to TDC_{ASD}. However, both disorders had almost identical total corpus callosum volume, in contrast to their controls in older years ($p = 0.96$ for ADHD in contrast to TDC_{ADHD} and $p = 0.95$ for ASD in contrast to TDC_{ASD}).

The analysis of the individual regions revealed that children with ADHD showed, in contrast to $TDC_{ADHD,}$ a significantly increased corpus callosum volume throughout all ages in the central ($p = 0.048$) and the mid-anterior ($p = 0.01$) region, and that these differences were mainly driven by differences in younger years ($p = 0.023$ for the central and $p = 0.004$ for the mid-anterior region).

3.5 Analysis of subcortical ROI volumes

For the analysis of subcortical ROI volumes, the following covariates were used: gender, age, IQ and TGMV. For a detailed report of these findings, refer to *Supplementary B7*.

Children with ADHD showed, in contrast to children with ASD, a significantly reduced volume in the **nucleus accumbens area** (p = 1.8E-6), the **amygdala** (p = 0.007) and the **nucleus caudate** (p = 0.012). In the nucleus accumbens area, this effect was observable in younger (p = 3.0E-5), as well as in older children (p = 0.004). In the amygdala, the difference was mainly driven by older children (p = 0.011), and in the nucleus caudate, the difference was mainly driven by younger children (p = 0.004). Additionally, the nucleus accumbens area showed to be significantly increased in younger children with ASD, in contrast to TDC_{ASD}.

The **pallidum** showed a significant decrease in volume (p = 0.016) only in older children with ADHD, in contrast to TDC_{ADHD}, The **putamen** showed a significantly decreased volume (p = 0.012) only in younger children with ADHD, in contrast to TDC_{ADHD}.. The **hippocampus** showed a trend (p = 0.08) towards a decreased volume in ADHD, in contrast to ASD. This trend was driven by significant differences in older children (p = 0.029). The **thalamus proper** volume is significantly decreased in children with ASD, compared to TDC_{ASD} (p = 0.042), as well as compared to children with ADHD (p = 0.029).

3.6 Analysis of surface-based morphometric measurements

The following section shows the results of the vertex-wise analysis of surface-based morphometric cortical measurements, for each estimated contrast. Gender, age and IQ were used as covariates in all vertex-wise analysis models. Additionally, MCT was used as covariate in the analysis of cortical thickness, TCSA as covariate in the analysis of cortical surface area, and TGMV as covariate in the analysis of cortical volume.

Figures 5 to 7 show the inflated surface maps of each hemisphere (light gray = gyri, dark gray = sulci). The views depicted in each row of these figures are, from left to right: lateral view left and right hemisphere, medial view left and right hemisphere, posterior view right and left hemisphere, anterior view left and right hemisphere, ventral view left and right hemisphere and dorsal view left and right hemisphere. Each cluster shown on the surface map has a cluster threshold of $p < 0.05$. The color-coding inside those clusters represents the p-value of each given vertex. P-values colored in red to yellow show a positive contrast effect, and p-values colored in blue to turquoise show a negative contrast effect. Section **A** in each

Figure reports the findings of *All ages*, section **B** of *Age 8 to 12* and section **C** of *Age 13 to 18*. In the following sections, the three cortical measurements thickness, surface area and volume are reported separately and each section reports all estimated contrasts separately. *Figure a* shows the results of the F-contrast, comparing all four groups. *Figure b* shows the results of the T-contrast, comparing ADHD to TDC_{ADHD}. *Figure c* shows the results of the T-contrast, comparing ASD to TDC_{ASD}. *Figure d* shows the results of the T-contrast, comparing ASD to ADHD. *Figure e* shows the results of the T-contrast, comparing ASD and ADHD to TDC.

For a more detailed description of the vertex-wise analysis outputs on the cortical thickness, cortical surface area and cortical volume shown in *Figures 5-7*, see *Supplementary B8*, *B9*, *B10* and *B11* respectively.

3.6.1 Analysis of the cortical thickness

F-contrast comparing all four groups

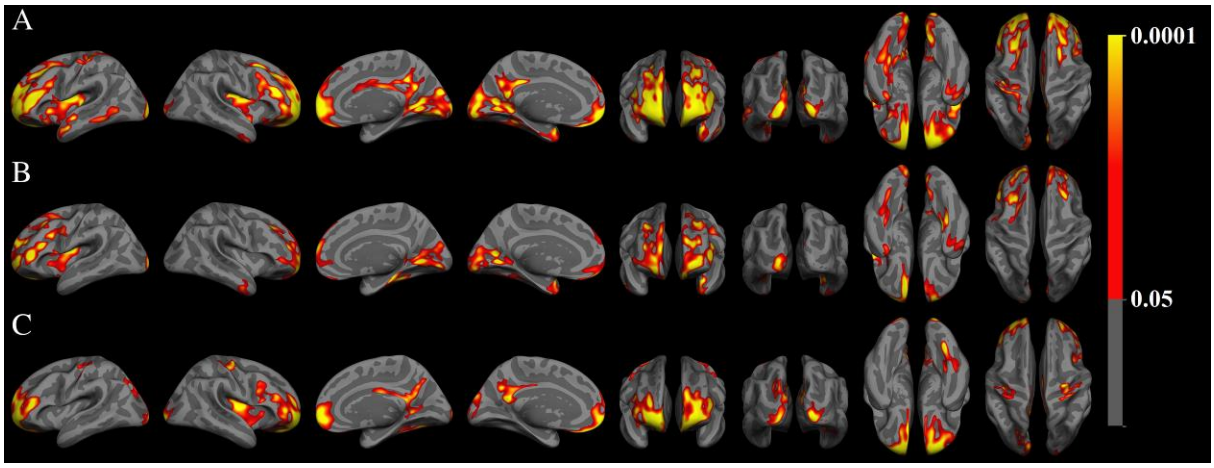

Figure 5a. *F-contrast comparing the cortical thickness between all four groups. For more information see Supplementary B8a.*[*]

The F-contrast comparing the cortical thickness between the four groups revealed effects in bilateral pre- and postcentral, rostral middle, caudal middle and superior frontal, frontal pole, medial and lateral orbitofrontal, pars orbitalis, pars opercularis, insula, middle and inferior temporal, fusiform, lateral occipital, lingual, pericalcarine, cuneus, isthmus cingulate, precuneus, in left superior parietal, superior temporal, temporal pole, parahippocampal, occipital pole, superior parietal and in right pars triangularis, corpus callosum, caudal anterior and posterior cingulate regions (A). The F-contrast comparing the cortical thickness between younger children revealed effects in bilateral superior and rostral middle frontal, medial and lateral orbitofrontal, cuneus, pericalcarine, lingual and precuneus, in left precentral, caudal

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

middle frontal, pars opercularis, pars triangularis, frontal pole, insula, postcentral, temporal pole, entorhinal, fusiform, occipital pole, lateral occipital and in right isthmus cingulate, inferior and middle temporal regions (B). The F-contrast comparing the cortical thickness between older children revealed effects in bilateral post- and precentral, occipital pole, precuneus, isthmus cingulate, superior and rostral middle frontal, frontal pole, lateral and medial orbitofrontal, in left inferior and superior parietal, cuneus, posterior cingulate and in right corpus callosum, lingual, lateral occipital, fusiform, insula, caudal middle frontal, pars opercularis and pars orbitalis regions (C).

T-contrast comparing ADHD to TDC_{ADHD}

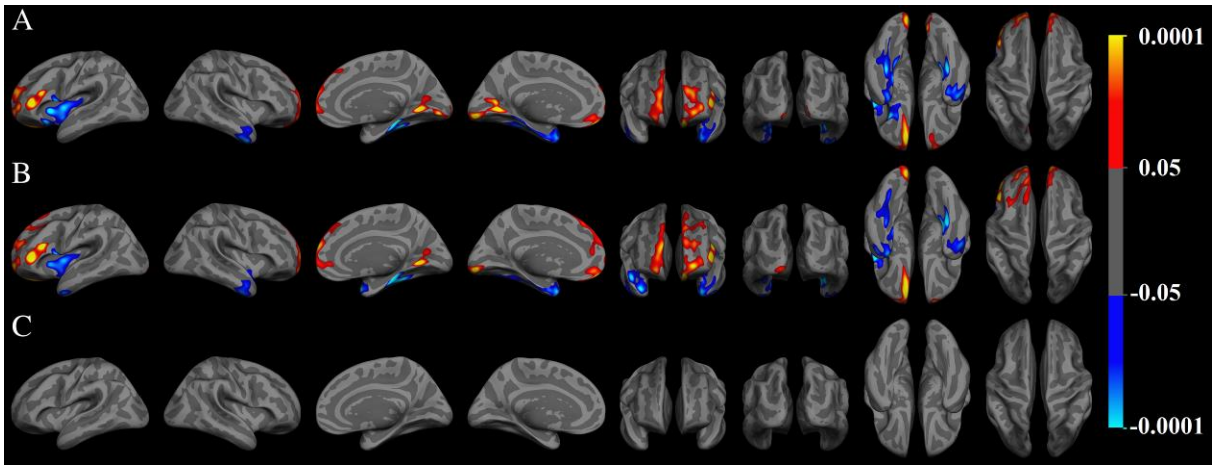

Figure 5b. *T-contrast comparing the cortical thickness of ADHD to TDC_{ADHD}. For more information see Supplementary B8b.*[*]

Children with ADHD showed, in contrast to $TDC_{ADHD,}$ an increased cortical thickness in bilateral lingual, pericalcarine, lateral orbitofrontal, superior and rostral middle frontal and in left medial orbitofrontal regions, as well as a decreased cortical thickness in bilateral fusiform, in left pars opercularis, precentral, insular, superior temporal, entorhinal, parahippocampal, and in right inferior and middle temporal regions (A). Younger children with ADHD showed increased cortical thickness in bilateral lingual, rostral middle frontal, medial orbitofrontal, superior frontal, frontal pole, in left lateral orbitofrontal, caudal middle frontal, pars triangularis, lateral occipital, and in right pericalcarine regions, as well as decreased cortical thickness in bilateral fusiform, parahippocampal, inferior and superior temporal, temporal pole, in left pars opercularis, precentral, entorhinal, temporal pole, and in right insular and middle temporal regions (B). Older children with ADHD showed no significant abnormalities in cortical thickness (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

T-contrast comparing ASD to TDC_{ASD}

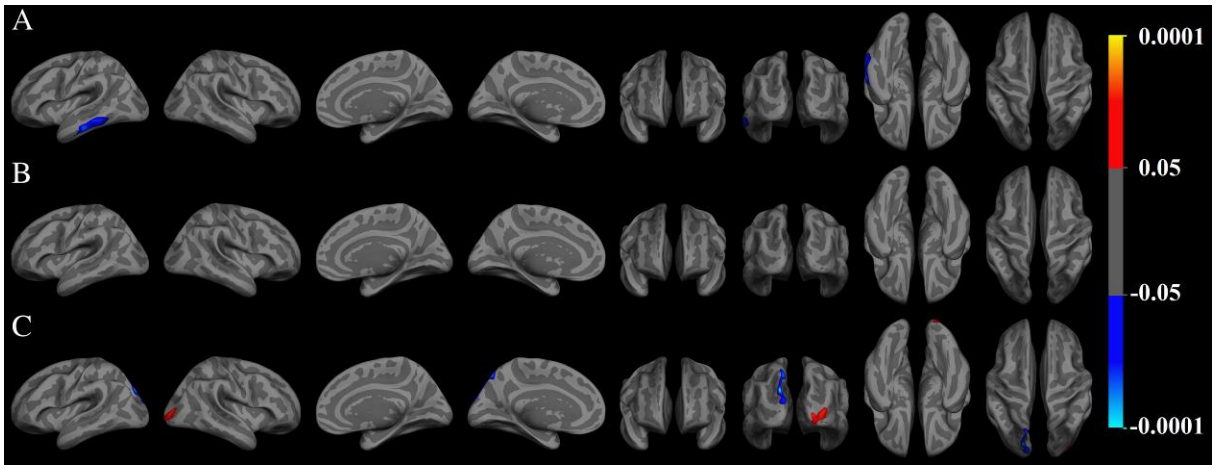

Figure 5c. *T-contrast comparing the cortical thickness of ASD to TDC_{ASD}. For more information see Supplementary B8c.*[*]

Children with ASD showed, in contrast to TDC_{ASD}, a decreased cortical thickness in left middle and inferior temporal regions (A). Younger children with ASD showed no significant abnormalities in cortical thickness (B). Older children with ASD showed an increased cortical thickness in right lateral occipital regions and a decreased cortical thickness in left superior parietal regions (C).

T-contrast comparing ASD to ADHD

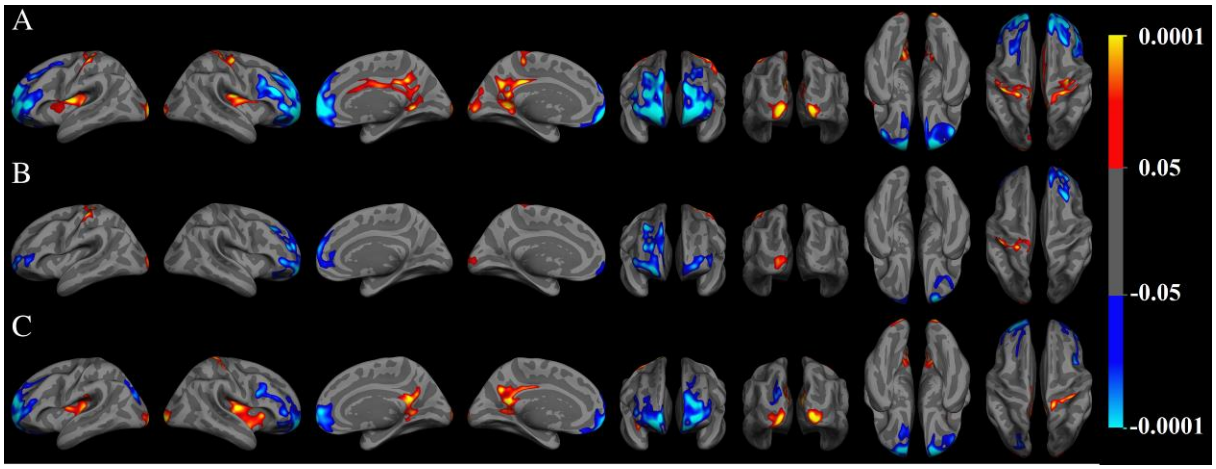

Figure 5d. *T-contrast comparing the cortical thickness of ASD to ADHD. For more information see Supplementary B8d.*[*]

Children with ASD showed, in contrast to children with ADHD, an increased cortical thickness in bilateral insula, post- and precentral, lateral occipital, precuneus, posterior- and isthmus cingulate regions, left paracentral, pericalcarine, cuneus and in right lingual, caudal anterior cingulate and corpus callosum, as well as a decreased cortical thickness in bilateral medial and lateral orbitofrontal, rostral middle, caudal middle and superior frontal, pars or-

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

bitalis and frontal pole and in right pars opercularis regions (A). Younger children with ASD showed, in contrast to children with ADHD, an increased cortical thickness in left post- and precentral and lateral occipital regions, as well as a decreased cortical thickness in bilateral medial orbitofrontal, rostral middle frontal, in left frontal pole and in right lateral orbitofrontal and superior frontal regions (B). Older children with ASD showed, in contrast to children with ADHD, an increased cortical thickness in bilateral insula, postcentral, lateral occipital, precuneus, isthmus cingulate, in left supramarginal and posterior cingulate regions, as well as a decreased cortical thickness in bilateral medial and lateral orbitofrontal, rostral middle and superior frontal, frontal pole, in left superior parietal and in right caudal middle frontal, pars opercularis and pars orbitalis regions (C).

T-contrast comparing ASD and ADHD to TDC

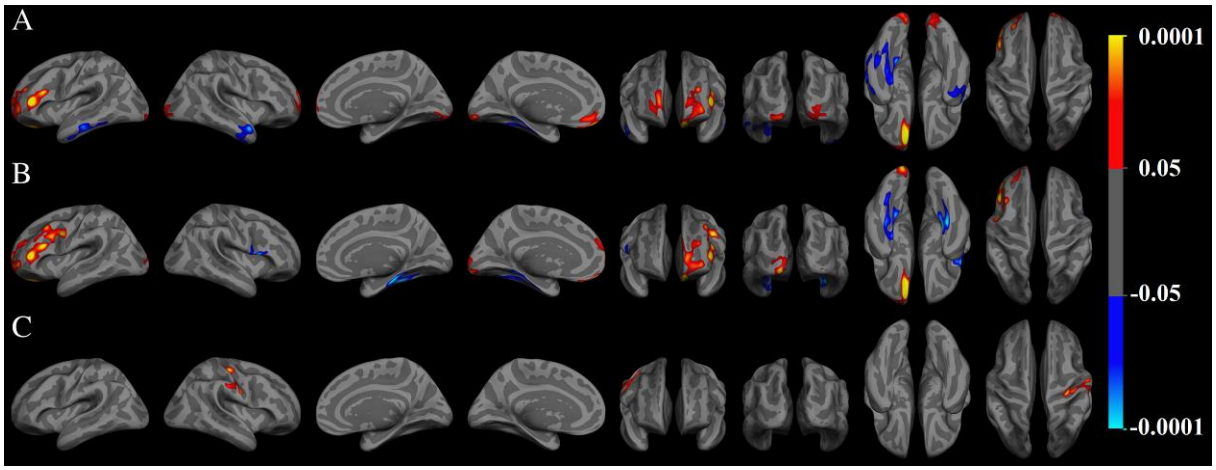

Figure 5e. *T-contrast comparing the cortical thickness of ADHD and ASD combined to TDC. For more information see Supplementary B8e.*[*]

Children with ADHD or ASD showed, in contrast to TDC, an increased cortical thickness in bilateral lingual, lateral occipital and rostral middle frontal, in left pars triangularis, medial and lateral orbitofrontal and in right superior frontal regions, as well as a decreased cortical thickness in bilateral middle and inferior temporal, in left fusiform, parahippocampal and in right superior temporal regions (A). Younger children showed an increased cortical thickness in left lateral occipital, occipital pole, medial and lateral orbitofrontal, rostral middle, caudal middle and superior frontal, par triangularis and precentral regions, as well as a decreased cortical thickness in bilateral fusiform and parahippocampal and in left pars opercularis and precentral regions (B). Older children showed an increased cortical thickness in right postcentral and precentral regions (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

3.6.2 Analysis of the cortical surface area

F-contrast comparing all four groups

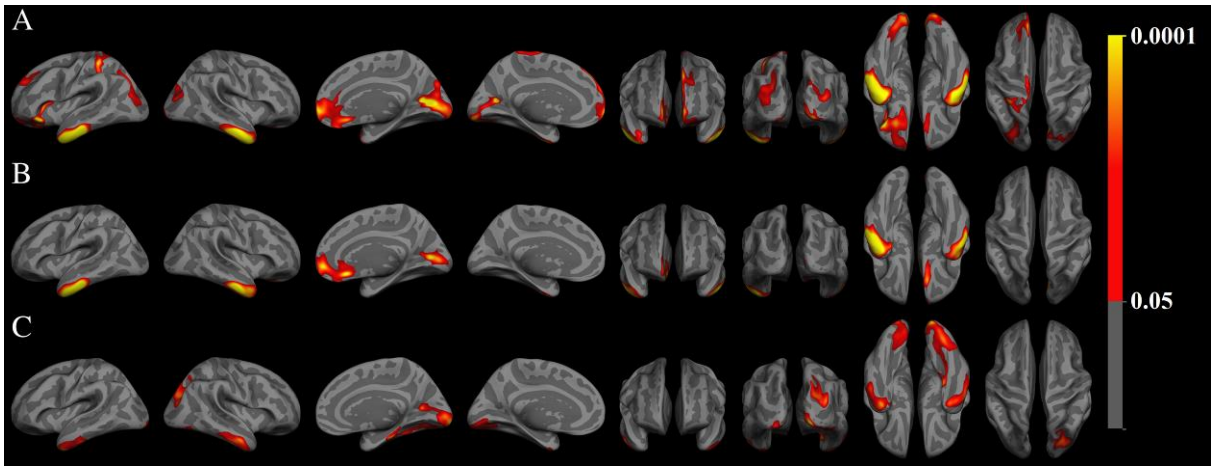

Figure 6a. *F-contrast comparing the cortical surface area between all four groups. For more information see Supplementary B9a.*[*]

The F-contrast comparing the cortical surface area between the four groups revealed effects in bilateral middle and inferior temporal, lateral occipital, pericalcarine, precuneus, inferior parietal, superior frontal, frontal pole, medial orbitofrontal, in left fusiform, entorhinal, lingual, para- and precentral, lateral orbitofrontal, pars orbitalis, pars triangularis and in right cuneus, superior parietal, superior temporal and rostral anterior cingulate regions (A). The F-contrast comparing the cortical surface area between younger children revealed effects in bilateral middle and inferior temporal, in left fusiform, entorhinal and right pericalcarine, rostral anterior cingulate, superior, medial and lateral orbitofrontal regions (B). The F-contrast comparing the cortical surface area between older children revealed effects in bilateral middle and inferior temporal, fusiform, lingual, lateral occipital and in right superior and inferior parietal, occipital pole, pericalcarine and parahippocampal regions (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

T-contrast comparing ADHD to TDC_{ADHD}

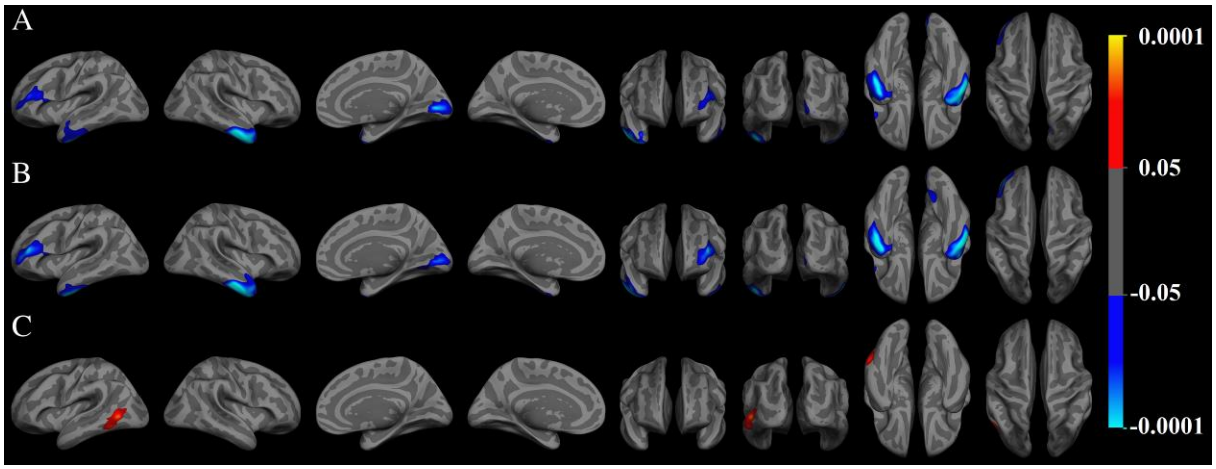

Figure 6b. *T-contrast comparing the cortical surface area of ADHD to TDC_{ADHD}. For more information see Supplementary B9b.*[*]

Children with ADHD showed, in contrast to $TDC_{ADHD,}$ a decreased cortical surface area in bilateral fusiform, middle and inferior temporal, in left rostral middle frontal, pars opercularis and in right pericalcarine, lingual, cuneus, temporal pole and superior temporal regions (A). Younger children with ADHD showed a decreased cortical surface area in bilateral middle and inferior temporal, in left fusiform, rostral middle frontal, pars opercularis and in right pericalcarine, lingual and superior temporal regions (B). Older children with ADHD showed an increased cortical surface area in left inferior and middle temporal regions (C).

T-contrast comparing ASD to TDC_{ASD}

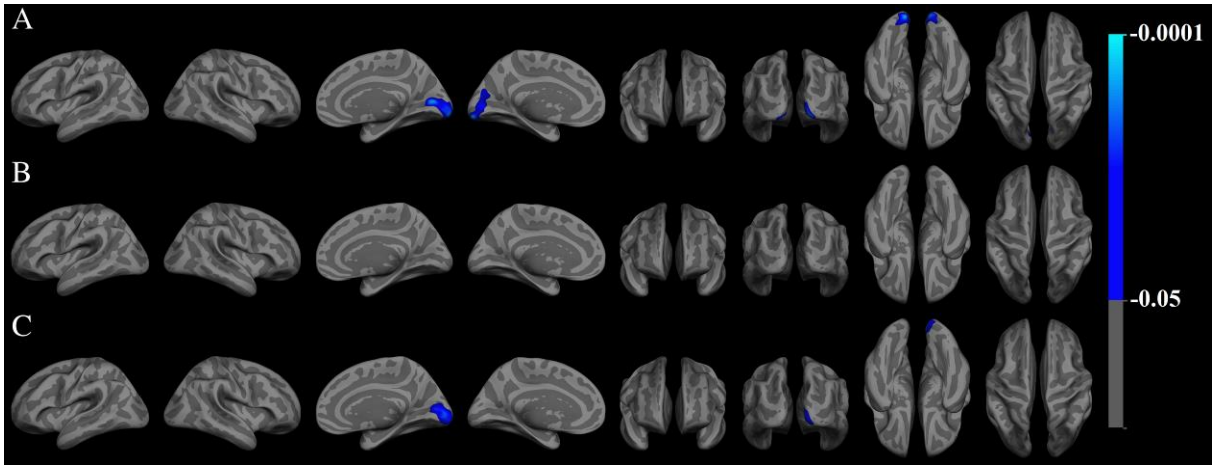

Figure 6c. *T-contrast comparing the cortical surface area of ASD to TDC_{ASD}. For more information see Supplementary B9c.*[*]

Children with ASD showed, in contrast to $TDC_{ASD,}$ a decreased cortical surface area in bilateral lingual, pericalcarine and lateral occipital and in left cuneus regions (A). Younger

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

children with ASD showed no significant abnormalities in cortical surface area (B). Older children with ASD showed a decreased cortical surface area in left lingual, pericalcarine, lateral occipital and cuneus regions (C).

T-contrast comparing ASD to ADHD

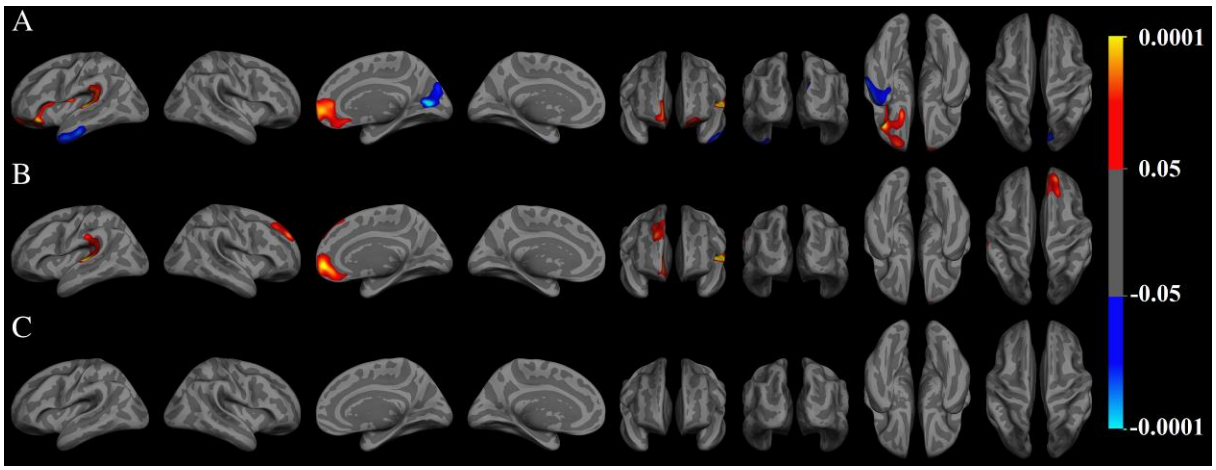

Figure 6d. *T-contrast comparing the cortical surface area of ASD to ADHD. For more information see Supplementary B9d.*[*]

Children with ASD showed, in contrast to children with ADHD, an increased cortical surface area in left superior temporal, supramarginal, insula, pars triangularis, pars orbitalis, lateral orbitofrontal and in right superior frontal, medial orbitofrontal and frontal pole regions, as well as a decreased cortical surface area in left middle and inferior temporal, and in right pericalcarine and cuneus regions (A). Younger children with ASD showed, in contrast to children with ADHD, an increased cortical surface area in left superior and transverse temporal, supramarginal and in right superior frontal, medial orbitofrontal and frontal pole regions (B). Older children with ASD showed no significant differences in cortical surface area in contrast to children with ADHD (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

T-contrast comparing ASD and ADHD to TDC

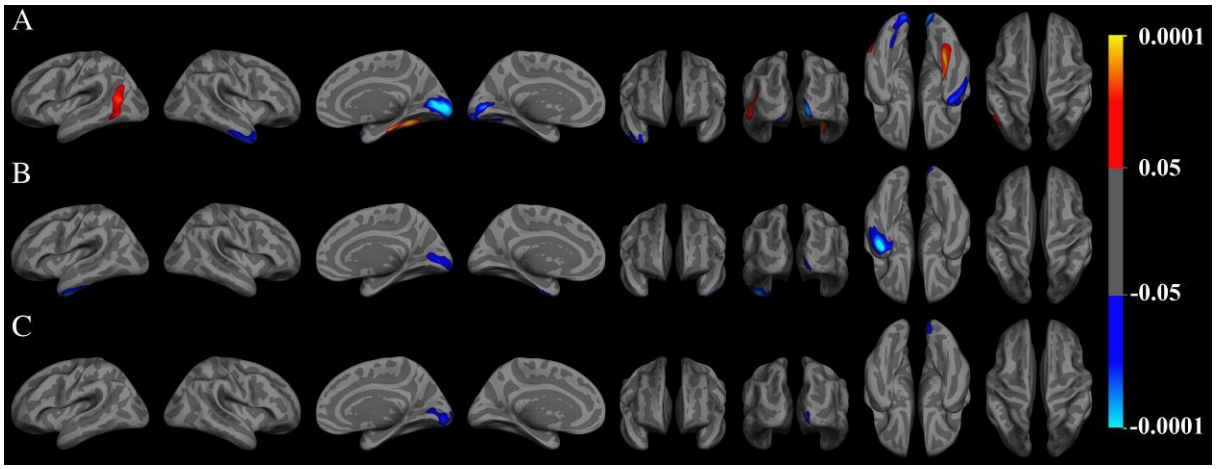

Figure 6e. *T-contrast comparing the cortical surface area of ADHD and ASD combined to TDC. For more information see Supplementary B9e.*[*]

Children with ADHD or ASD showed, in contrast to TDC, an increased cortical surface area in left inferior parietal, middle and inferior temporal and in right fusiform and parahippocampal regions, as well as a decreased cortical surface area in bilateral lingual, pericalcarine, in left lateral occipital and in right cuneus, temporal pole, inferior and middle temporal regions (A). Younger children with ADHD or ASD showed a decreased cortical surface area in left inferior temporal, fusiform, entorhinal and in right pericalcarine regions (B). Older children with ADHD or ASD showed a decreased cortical surface area in right pericalcarine and lingual regions (C).

3.6.3 Analysis of the cortical volume

F-contrast comparing all four groups

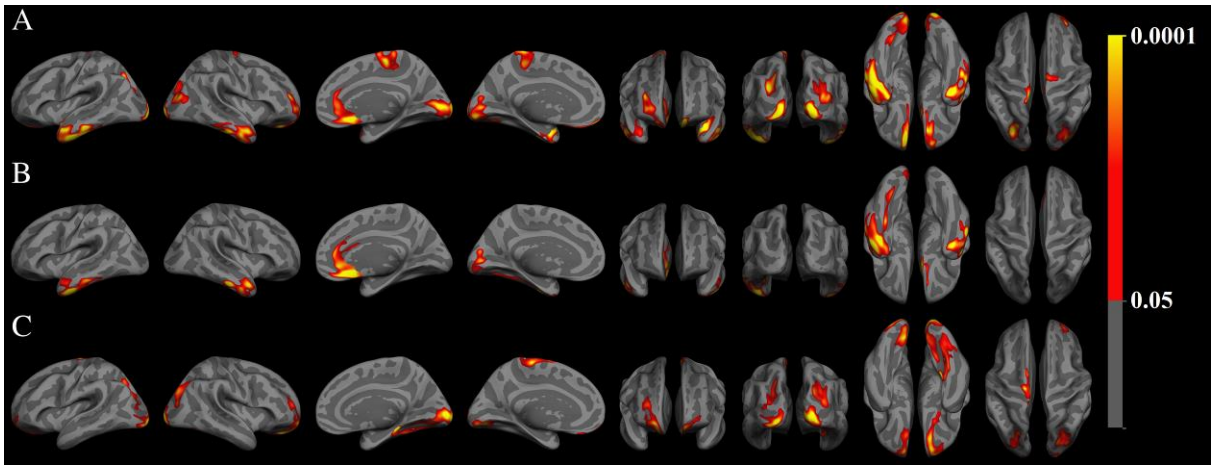

Figure 7a. *F-contrast comparing the cortical volume between all four groups. For more information see Supplementary B10a.*[*]

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

The F-contrast comparing the cortical volume between the four groups revealed effects in bilateral superior, middle and inferior temporal, fusiform, lingual, pericalcarine, lateral occipital, occipital pole, inferior and superior parietal, paracentral, precentral, medial and lateral orbitofrontal, in left entorhinal, insula, cuneus, postcentral, frontal pole and in right rostral middle frontal and rostral anterior cingulate regions (A). The F-contrast comparing the cortical volume between younger children revealed effects in bilateral middle and inferior temporal, fusiform, in left lingual, pericalcarine, cuneus and in right medial orbitofrontal and rostral anterior cingulate regions (B). The F-contrast comparing the cortical volume between older children revealed effects in bilateral lateral orbitofrontal, frontal pole, rostral middle frontal, lingual, lateral occipital, occipital pole, superior and inferior parietal, in left paracentral, precentral, superior frontal, medial orbitofrontal and in right pericalcarine, fusiform and parahippocampal regions (C).

T-contrast comparing ADHD to TDC_{ADHD}

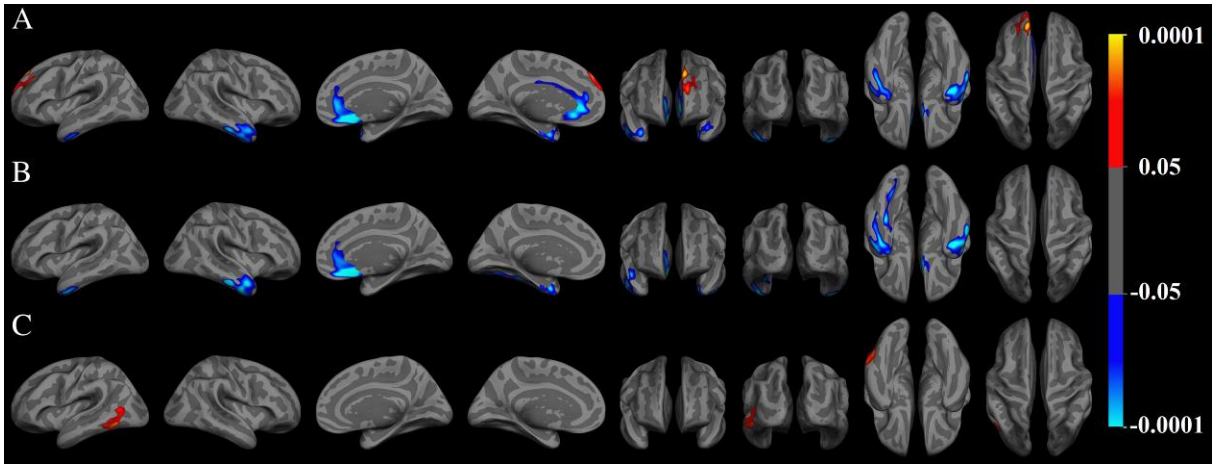

Figure 7b. *T-contrast comparing the cortical volume of ADHD to TDC_{ADHD}. For more information see Supplementary B10b.*[*]

Children with ADHD showed, in contrast to $TDC_{ADHD,}$ an increased cortical volume in the left superior frontal region, as well as a decreased cortical volume in bilateral rostral anterior cingulate, fusiform, inferior and superior temporal, in left entorhinal, caudal anterior cingulate and in right medial orbitofrontal and middle temporal regions (A). Younger children with ADHD showed a decreased cortical volume in bilateral inferior temporal and fusiform, in left entorhinal, temporal pole, and in right superior and middle temporal, medial orbitofrontal and rostral anterior cingulate regions (B). Older children with ADHD showed an increased cortical volume in the left inferior temporal region (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

T-contrast comparing ASD to TDC_{ASD}

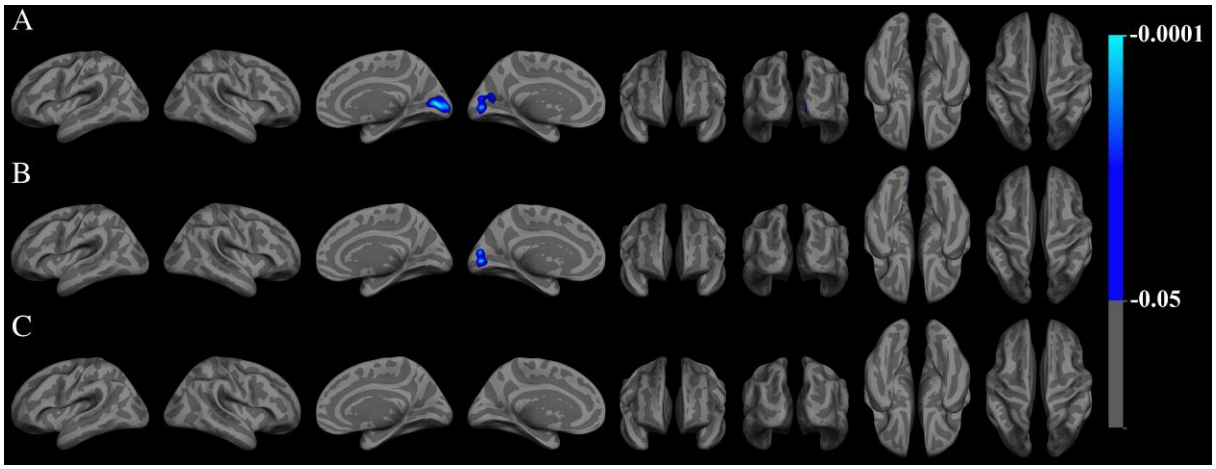

Figure 7c. *T-contrast comparing the cortical volume of ASD to TDC_{ASD}. For more information see Supplementary B10c.*[*]

Children with ASD showed, in contrast to $TDC_{ASD,}$ a decreased cortical volume in bilateral lingual and pericalcarine regions (A). Younger children with ASD showed a decreased cortical volume in left pericalcarine and cuneus regions (B). Older children with ASD showed no significant abnormalities in cortical volume (B).

T-contrast comparing ASD to ADHD

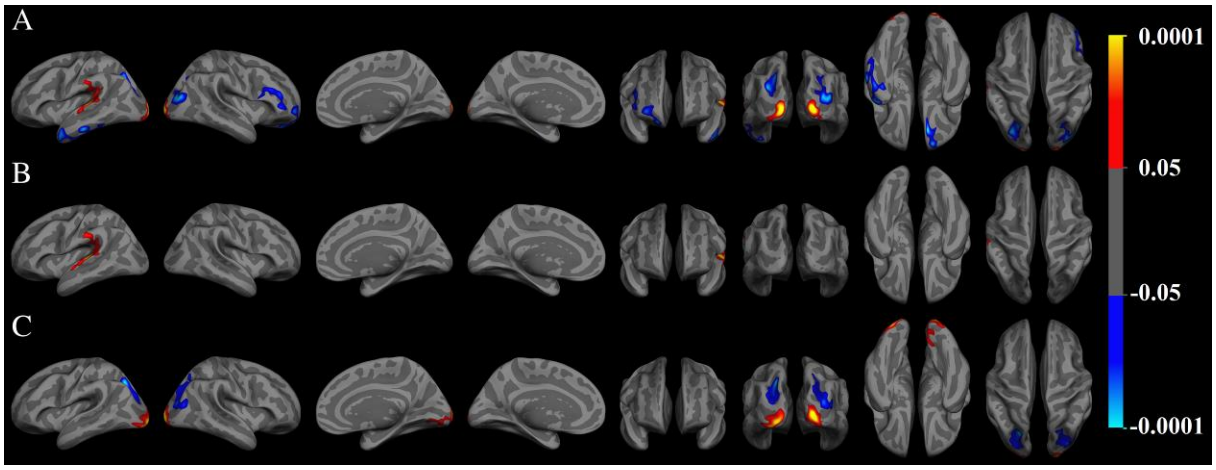

Figure 7d. *T-contrast comparing the cortical volume of ASD to ADHD. For more information see Supplementary B10d.*[*]

Children with ASD showed, in contrast to children with ADHD, an increased cortical volume in left superior temporal, supramarginal, lateral occipital and in right occipital pole regions, as well as a decreased cortical volume in bilateral inferior and superior parietal, in left middle and inferior temporal and in right lateral occipital, pars opercularis, rostral middle frontal and lateral orbitofrontal regions (A). Younger children with ASD showed, in contrast

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

to children with ADHD, an increased cortical volume in left supramarginal and superior temporal regions (B). Older children with ASD showed, in contrast to children with ADHD, an increased cortical volume in bilateral lateral occipital, occipital pole and in right lingual regions, as well as a decreased cortical volume in bilateral superior and inferior parietal regions (C).

T-contrast comparing ASD and ADHD to TDC

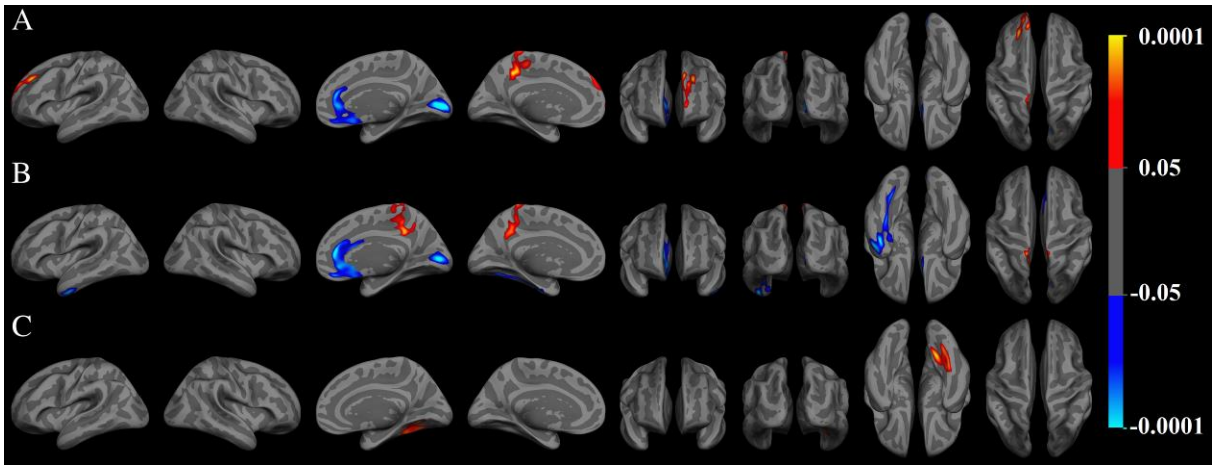

Figure 7e. *T-contrast comparing the cortical volume of ADHD and ASD combined to TDC. For more information see Supplementary B10e.*[*]

Children with ADHD or ASD showed, in contrast to TDC, an increased cortical volume in left precuneus, paracentral, superior frontal and rostral middle frontal regions, as well as a decreased cortical volume in right lingual, medial orbitofrontal and rostral anterior cingulate regions (A). Younger children with ADHD or ASD showed an increased cortical volume in bilateral paracentral, precuneus and in left postcentral regions, as well as a decreased cortical volume in left inferior temporal, fusiform, entorhinal and in right pericalcarine, medial orbitofrontal, rostral and caudal anterior cingulate regions (B). Older children with ADHD or ASD showed an increased cortical volume in left lingual and fusiform regions (C).

[*] Explanation to the depicted images in this figure can be found in the second paragraph of section 3.6.

4. Discussion

This study aimed at identifying structural similarities and differences between ADHD and ASD children's brain. It further assessed the differences between the brain of children with ADHD or ASD and TDC. While some findings postulated by previous literature could be reproduced, others were not observed. The following sections will first summarize group differences in age, IQ and gender, will then list global and local brain measurements and relate them to the literature. Finally, the limitations of this study will be given and concluding remarks, as well as recommendations for a future approach, will be stated.

4.1 Age, IQ and gender

The group of children with ADHD was significantly younger than the group of children with ASD. This age difference complicates the comparison between the two disorders and the interpretation of results, as significant findings might be better explained by age variation than by group differences. But this confounding factor was accounted for in the analyses, as age was always used as covariate of no interest.

Both ASD and ADHD groups showed significantly decreased IQ in comparison to their controls, but not to one another. This is in line with previous studies reporting IQ differences between ADHD and normally developing children (Castellanos et al., 1996).

Finally, a gender difference was found between ADHD children and their controls. This is in line with previous literature reporting a higher incidence of ADHD in male than in female gender (Levi et al., 2005). As opposed to the ADHD group, the ASD group showed no significant effect of gender distribution compared to its control group. Although literature reports a higher incidence of ASD in males (Kim et al., 2011), it can be that the ASD group involved in this study was better controlled for gender differences than the ADHD group.

4.2 Global brain measurements

As reported by previous literature, children with ADHD showed a significantly smaller TGMV than TDC and ASD (Valera et al., 2007). Further, the lack of significance of this effect in the sample of older children could be related to a disappearance of this decreased brain volume in ADHD with age. This contrasts with findings of a longitudinal study that reported a decreased global brain volume persisting into adulthood (Castellanos et al., 2002a).

The differences in TGMV in ADHD seems to be mainly driven by differences in TCSA and not by differences in MCT, as the differences between ADHD and TDC_{ADHD} seem to co-

variate in the same way in TGMV and TCSA measurements, but not in MCT. That is, both disorder groups show a normal growth trajectory concerning the thickness of the cortex. Thus, most findings of abnormal brain volume in either disorder (see *Table 2*) are actually based on differences in cortical surface area. To further investigate the cause of these detected cortical volume differences, future studies should additionally focus on cortical folding measurements. Indeed, the analysis of local cortical folding could give further insights on how to detect and interpret the consequence of an increased surface area. Further, if the local volume and thickness did not show any abnormalities, a local increase in surface area could only be explained by an increase in local cortical folding.

The expected growth trajectory of brain volume in ASD, which was described as an increased brain volume in younger children and a plateauing effect in older children where the brain volume converges to the one of normal children (Courchesne et al., 2007), could not be observed in the current sample of children, as TGMV did not differ between ASD and TDC_{ASD} at any time point. The lack of finding of an enlarged total brain volume in ASD compared to TDC_{ASD} could be explained by the findings of Courchesne et al. (2001). They reported that solely ASD children aged of 2 to 3 years showed an enlarged brain, and that this effect was not detectable anymore in older children aged of 5 to 16 years. Taken altogether, this suggests that the current group of children with ASD was already too old to detect the suggested abnormal brain growth in ASD.

There is a general uncertainty about the influence that white matter volume abnormalities have on the total brain enlargement. Some studies report a significant increased WM volume in ADHD (Narr et al., 2009), while others find a significant decrease in WM volume (Castellanos et al., 2002a), or no differences at all (Carmona et al., 2005). As the present study did not reveal any global differences in total white matter volume between any groups, it could indicate that the main reason for total brain volume differences in ADHD is based on abnormalities in gray matter volume.

Concerning the gray matter to white matter ratio, only older children with ASD showed a significant difference to TDC_{ASD}. This interesting finding supports the hypothesis of a more localized and hyper-connected brain in ASD (Markram & Markram, 2010). Assuming that changes in the efficiency of long distance connections, either more connections or thicker axonal diameter of connections, can be seen mostly in changes in white matter regions of sMRI scans and if changes in local connections between and within cortical minicolumns are represented by changes in gray matter regions of sMRI scans, then changes in the gray to white matter ratio might represent the ratio between short to long distance connections. In younger

children of any group (ADHD, ASD & TDC) the ratio between GM and WM is rather high with a value above 1.5, which probably represents the cortical overgrowth in connections found in any, learning and developing young child. In older children, synaptic pruning takes place leading to the development of more efficient cortical networks. While important long-range connections are held intact and unnecessary short-range connections are pruned, the GM to WM ratio should decreases with age. This necessary decrease of GM to WM ratio seems to be significantly slower in older children with ASD than in older TDC_{ASD}. This could be due to a more localized and hyper-connected autistic brain and would support the intense world theory postulated by Markram & Markram (2010), stressing the possibility of high interconnection of local neural microcircuits. Additionally, it supports the assumption that ASD might rather be a disorder with a disturbed growth trajectory than an abnormal growth outcome as postulated by Amaral et al. (2008). In contrast, ADHD seems to be a disorder with an abnormal growth outcome, which is mainly based on a delayed growth rate as differences between ADHD and TDC_{ADHD} seem to be most pronounced in younger years (see *Supplementary B11*).

The analysis of cerebellar GM or WM volume showed no significant differences between any groups of interest. The cause of the lack of replication of previous findings in ASD (Amaral et al., 2008) and in ADHD (Castellanos et al., 2002a) remains unclear. However, this could be due to the fact that other studies focused more strongly on specific cerebellar regions such as the cerebellar vermis (Valera et al., 2007) and did not look at total cerebellar GM or WM volume. In addition, this divergence from the preexisting literature could be due to the lack of control for TGMV, in contrast to the report at hand, which accounted for it. Further analysis could provide additional insight on this matter.

Differences in corpus callosum volume between either ASD or ADHD and their controls could not be observed. However, there is a trend of generally increased corpus callosum volume in younger children of either disorder. This strongly contrasts with previous studies, reporting general decreased corpus callosum volume in children with ASD or ADHD (Hardan et al., 2000; Hutchinson et al., 2008). The absence of volume differences in the corpus callosum between ASD or ADHD and TDC seems to be a valid argument against abnormal long distance connections in either disorders and therefore challenges the postulated aberrant connectivity theory. However, the discrepancy between the current findings and the literature might be due to the way the regional volume differences are estimated. In this study, the volume of the corpus callosum and its subparts was automatically segmented by FreeSurfer's segmentation algorithm, while the corpus callosum volume in the literature was mostly esti-

mated through manual or semi-manual segmentation. The question of the sensitivity and reliability of both methods has to be assessed in further studies. However, as the corpus callosum consists of white matter fiber tracts, the best approach to accurately measure corpus callosum properties would probably be the use of DTI.

4.3 Local brain measurements

The analysis of localized effects in cortical thickness, surface area or volume in both disorders revealed interesting findings. A summary of these findings can be found in *Supplementary B11*. *First*, similarities and differences between the current findings and the literature are pointed out. *Second*, regions that are specific to one or the other disorder are reported. *Third*, regions that distinguish the two disorders from each other are presented and *fourth*, regions that are commonly abnormal in both disorders, in contrast to TDC, are reported.

4.3.1 Replication of previous findings

Children with ASD showed, in contrast to TDC_{ASD}, a decreased volume in the occipital lobe (pericalcarine and lingual cortex) and the thalamus, which is in line with previous findings (Brieber et al., 2007; Tsatsanis et al., 2003). Concerning the inconsistent finding of an increased (Hardan et al., 2006b) or a decreased (Hardan et al., 2009) cortical thickness in the temporal lobe in previous literature, my results would support a general decreased cortical thickness in temporal regions, especially in the left hemisphere. New insights are provided by the analysis of cortical surface area, which shows that children with ASD have a generally increased frontal lobe surface area (mostly seen in younger children) and a decreased occipital lobe surface area (mostly seen in older children). Even though the literature reveals many more specific regional modulations in ASD (see *Table 2*), solely the previously mentioned results were found in the current study. The concurrent occurrence of occipital volume and surface area decrease, without changes in thickness, suggests that local occipital volume abnormalities are mainly driven by surface area effects.

Children with ADHD showed, in contrast to TDC_{ADHD}, a decreased cortical volume in medial orbitofrontal and anterior cingulate regions and throughout the temporal lobe, a decreased cortical thickness in the temporal lobe and a decreased cortical surface area in the occipital and temporal lobe. These findings are in line with previous studies (Carmona et al., 2005; Castellanos et al., 2002a; Shaw et al., 2007; Wolosin et al., 2009). However, the findings of an increased cortical volume in children with ADHD in left superior frontal regions,

an increased volume in corpus callosum (mainly in central and mid-anterior regions), an increased cortical surface area in the parietal lobe and a local decreased cortical volume in the pars opercularis and precentral regions, are contrasting with the existing literature (Gargaro et al., 2011; Seidman et al., 2005; Wolosin et al., 2009). Finally, this study revealed differences that were not previously reported in the literature, such as the one found in the occipital lobe, which was an increased cortical thickness, especially in pericalcarine and lingual regions, as well as the one in the insular cortex, which showed a general decreased volume. In addition, the finding of an increased cortical thickness throughout the frontal lobe, so far unreported in literature, can be explained by findings reported by Shaw et al. (2007), who stressed that children with ADHD reach the peak of maximal cortical thickness at 10.5 years, while TDC reach their peak earlier at 7.5 years. Further, this effect is most prominent in prefrontal regions. This could support the current findings, as the group of young children contains children between the ages of 8 and 12 years. This could also explain why the cortical thickness difference between ADHD and TDC_{ADHD} was not present anymore in the group of older children.

Children with ASD showed, in contrast to ADHD, a decreased volume in frontal and parietal regions and in the thalamus, an increased volume in the occipital and cingulate cortex and in many subcortical regions, such as the nucleus accumbens, amygdala, caudate nucleus, putamen and hippocampus. While the increased volume in subcortical regions is supported by the literature (see *Table 2*), the decreased volume in frontal and parietal regions is in contrast to previous findings, which showed rather increased volume in these areas in ASD and decreased volume in ADHD. The increased brain volume in ADHD could be explained by a delayed synaptic pruning process in the frontal cortex. This could occur because the neural network in children with ADHD is not yet fully established, while a functioning, albeit abnormal wired, neural network is already built in children with ASD. A longitudinal study with well-matched age groups that also records DTI measurements would help to further establish the cause of this observation and assess whether the general decreased frontal and parietal cortical volume is solely due to the age difference between the groups or if it actually represents disorder specific differences. Children with ASD also showed, in contrast to ADHD, a decreased cortical thickness in many frontal lobe regions and an increased cortical thickness in para-, pre- and postcentral regions, the precuneus, and in the occipital, the cingulate, and the insular cortex. The increased cortical thickness around the central sulcus and in the occipital, cingulate and insular cortex could be caused by an increase of local connections within minicolumns, and might explain the symptoms of restricted, repetitive patterns of behavior (APA, 2013), as well as a more fragmented visual perception of objects in children with ASD (Mar-

kram & Markram, 2010). Additionally to the differences in volume and thickness, children with ASD also showed, in contrast to children with ADHD, a regional decrease in cortical surface area in parietal and occipital lobe, as well as an increased surface area in left insula, left superior temporal, left supramarginal regions and in many regions of the frontal lobe.

Children with ASD or ADHD showed, in contrast to TDC, an increased cortical volume in some regions of the frontal and parietal lobe and a decrease in cortical volume in some regions of the temporal and limbic lobe. The findings of a generally increased parietal lobe and a decreased temporal lobe are consistent with previous literature (Brieber et al., 2007), while the finding of an increased frontal volume was not expected, as children with ADHD are rather known to have a decreased frontal volume, while the frontal volume in ASD is increased (see *Table 2*). Further research is needed to assess whether this frontal effect is due to a delayed maturation process in neurodevelopmental disorders in general. The brain of children with either disorder seems to show, in general, an increased cortical thickness, as local increases were observed in many frontal and occipital lobe regions. Some regional decreases in thickness were solely found in fusiform and parahippocampal regions. Cortical surface area was decreased in children of either disorder in the occipital lobe, while the temporal lobe showed rather heterogeneous results. Many of the regions shown to be abnormal in both disorders, such as frontal, cingulate and parietal regions, as well as precuneus and parahippocampal regions, are part of the default mode network (Minshew & Keller, 2010). Future research of functional connectivity abnormalities in ADHD or ASD could provide further insight in that matter.

4.3.2 Regions specific to disorders

No clear region could be defined as typical for brain abnormalities in ASD. The only candidate nearly showing ASD-specific characteristics was the occipital lobe, which showed, in contrast to $TDC_{ASD,}$ a decrease in cortical surface area and volume.

The identification of disorder specific brain abnormalities was much easier for ADHD, as a general decreased total cortical volume and total surface area could be identified as ADHD specific. Further, this is supported by previous findings (Valera et al., 2007; Wolosin et al., 2009). Additionally, the temporal lobe seemed to be abnormal almost solely in ADHD as it showed a decreased volume, thickness and surface area in almost all these regions. Moreover, an increase in cortical thickness in most frontal and occipital regions was also ADHD specific.

It must be stressed that abnormalities in ADHD revealed in this study were almost exclusively observed in the sample of younger children and often disappeared in older subjects. In contrast, effects found in ASD were equally often supported by abnormalities in younger and older children (see *Supplementary B11*).

4.3.3 Regions differentiating disorders

The most characteristic differences between children with ASD and children with ADHD were the overall increased TGMV and MCT, the increased volume in subcortical regions, such as the nucleus accumbens, amygdala, caudate nucleus, putamen and hippocampus, the increased cortical thickness around the central sulcus and in the occipital, the cingulate and the insular cortex, the decreased cortical thickness in the frontal lobe, as well as the increased cortical surface area in the frontal lobe. According to Amaral et al. (2008), the regions mostly responsible for the neuropathology of social behavior are the frontal lobe, superior temporal cortex, the parietal cortex and the amygdala. All these areas showed abnormal increased measurements in ASD compared to ADHD, and therefore could account for the fact that children with ASD have more problems with social communication and interactions compared with children with ADHD. Further, this study enlightened that the orbitofrontal cortex, the caudate nucleus and the thalamus were abnormal in ASD compared to ADHD. These areas were reported to be associated to the neuropathology of repetitive or stereotyped behaviors, one of the main symptoms of autism (Amaral et al., 2008).

4.3.4 Regions unifying disorders

Only few regions showed similar abnormalities in both disorders. Most consistent are the findings of decreased volume in the cingulate cortex, increased thickness in the frontal lobe, decreased thickness in the temporal lobe, and decreased surface area in the occipital lobe. Both disorder showed an increase in cortical thickness throughout the orbitofrontal cortex, a region associated with social disinhibition and impulse control (Seidman et al., 2005). These findings would support the theory of executive dysfunction in both disorders. This is further supported by the fact that cortical thickness is significantly more increased in children with ADHD than in children with ASD, as children with ADHD have much more problems with disinhibition and impulse control. Yet, the reason for stronger impairment in some executive functions in one or the other disorder remains unclear. It could be explained by regions with disorder specific structural abnormalities or regions which show structural differences

between the disorders, as described by the previous two sections. The best approach to answer this question is probably the execution of well-designed fMRI paradigms, specifically aiming at distinguishing between the different degrees of impairment in executive functions in ASD or ADHD.

Additionally, the analysis of functional connectivity networks, acquired through rsfMRI scan, might help to further explore disorder specific abnormalities in functional networks, such as the default mode network (Minshew & Keller, 2010), the dorsal and ventral attentional network (Castellanos & Proal, 2012) and the salience network (Seeley et al., 2007). A hint of abnormal functional connectivity network in either disorder can already be seen in structural abnormalities in "rich club hubs" (van den Heuvel & Sporns, 2011), such as the precuneus, the superior frontal and parietal cortex and the thalamus. A stronger focus on regions that are important for executive functions, such as fronto-striatal structures, might help to understand the network intrinsic abnormalities that represent either ASD, ADHD or both.

4.4 Limitations

The aim of finding unifying and differentiating neuroanatomical abnormalities in both disorders ADHD and ASD, and this all over the brain is a notable endeavor; further, it is also prone to many errors and contains many limitations.

First, on the level of subjects: By looking at so many subjects at once, it was impossible to account for the whole consortium that causes interindividual variability, either because of missing information or of the small number of subjects within subcategories. One missing information example would be the factor of medication or treatment. It remains unclear whether the use of medication actually influences brain volume. While Castellanos et al. (2002a) found no GM volume differences between medicated and unmedicated children with ADHD, Nakao, Radua, Rubia and Mataix-Cols (2011) reported that the administration of medication in people with ADHD led to a more normal brain volume. Similarly for the treatment, Frodl and Skokauskas (2012) reported that with treatment and time, brain abnormalities in children with ADHD diminished as they become older, and that some brain abnormalities, such as an ACC volume reduction, were still detectable in untreated adults with ADHD. Further, an example for the too small number of subjects per subcategory would be the factor of gender. This study accounted for gender, but could not provide relevant information on gender differences. This is an issue, as the study by Bloss and Courchesne (2007) suggested gender specific differences in size-related white matter abnormalities in autistic children. The too small number of subjects per subgroup was also a drawback, as the two disorders could not be ana-

lyzed in respect to their subgroups ADHD-I, ADHD-C, ADHD-C, Asperger's and Autism. This is unfortunate, as some studies revealed a big difference between the different subtypes (Mayes et al., 2012). Geurts et al. (2004) reported that, in contrast to ADHD-H and ADHD-C, ADHD-I should not be associated with deficits in executive functions. It remains unclear whether ADHD-I does really belong to ADHD, or if it is a distinct attentional disorder by itself (Carmona et al., 2005). Further, others hypothesized that there is no clear distinction between ADHD-C and autism (Mayes et al., 2012).

Second, on the level of sites: The most obvious confounding factor in this study is the heterogeneity between the different datasets caused by subject specific characteristics or scanner specific parameters. There are many site-specific sources of variation that could account for any confounding or mediating factors, such as differences in behavioral measurement, diagnostic tools used, imaging data acquisition, protocol and scanner quality, technicians and experimenters involved, subject recruitment protocol, medication or treatment status of subjects, comorbidities, inclusion (e.g. IQ above 80 or 85, only autistic children or also children with Asperger's syndrome) and exclusion criteria (e.g. no fragile X syndrome, no affective disorder, no mental retardation, no seizures), policies and reasons for contributing data, handling of missing data (Eloyan et al., 2012; Schumann et al., 2004; Seidman et al., 2005). All these sources of scanner-specific heterogeneity can lead, in a big dataset, to the clustering of characteristics, a phenomenon called "batch effect" (Olivetti et al., 2012). Unfortunately, I was not able to account for this batch effect, as the present analysis algorithms were either not capable of accounting for multiple sources of data or if so, no significant results remained. Further connected to this limitation is the issue of group matching. To keep the number of subjects as high as possible, I did not match the different sites and group by factors such as gender, IQ and age. Indeed, the application of appropriate matching and randomization process would have highly decreased the total number of subjects and therefore increased the problematic of scanner-specific influential factors. Nonetheless, the overall distribution of gender and IQ in this study represents more or less the actual skewed distribution of those characteristics in the population. Solely the age difference between the group of children with ADHD and those with ASD is worrisome, as it weakens the direct comparison of ADHD to ASD.

Third, on the level of methodology: One of the main drawbacks in methodology is that this study postulated hypotheses about many different brain regions and is therefore prone to the problem of multiple comparisons. Briefly, if looking at a lot of things, something will forcibly be interesting and significantly different. In line, this study probably focuses on too

many factors at once. The depth of analysis might not be enough detailed. Many of the anatomical findings in the field showed a lateralized effect. By making no distinction between the two hemispheres, this study might lose the potential of finding hemisphere-specific abnormalities or disorder-specific asymmetries. Another methodological problem of this study is that I tried to infer the growth trajectory of neurodevelopmental disorders by looking at cross-sectional data. This is a fundamental flaw, as brain enlargement found in cross-sectional data does not have to imply real growth abnormality (Amaral et al., 2008). Nonetheless, this is a general and well known weakness in this field, as almost all findings mentioned in *Table 1* and *2* are from cross-sectional studies and therefore inherit the same flaw as the present study. Finally, there is also the unknown software variability of FreeSurfer's parcellation and segmentation algorithm and the dependencies of the used atlases. FreeSurfer's volumetric segmentation atlas, for example, is based on 39 people (28 healthy subjects and 11 patients with questionable or probable Alzheimer's disease) (Fischl et al., 2002) and the surface parcellation atlas is based on 40 people aged of 19-87 years (Desikan, et al., 2006). As the subjects in the current study were all much younger and unaffected by any aging-based dementia disease, it is unclear how well FreeSurfer's algorithms performed. As well described by Makris et al. (2007), the use of an automated segmentation procedure to map something as complex and highly curved as the neocortex increases the speed of analysis while simultaneously decreasing its accuracy. Such algorithms have problem accounting for the high degree of interindividual variability and are prone to registration errors. Still, the methods and algorithms used in this study represent the state-of-the-art technology in this domain.

Taken together, the lack of reproduction or the discrepancies compared to previous literature's findings could be explained by the fact that many previous studies did neither correct for the confounding influence of age, IQ, gender, nor total brain volume nor did these studies adjust for global TGMV, MCT, or TCSA when analyzing the respective local brain measurements.

Either of these limitations could account for the lack of finding of stronger reinforcement for the theory of executive dysfunction and the aberrant connectivity theory. While the previously mentioned disorder unifying regions helps to understand why ASD and ADHD have so many symptomatic overlaps, the disorder differentiating regions cannot fully explain the factors that separate the two disorders.

4.5 Conclusion and future approaches

The present study revealed that children with ADHD or ASD show abnormal structural brain differences in comparison to TDC or to each other. According to the postulated hypothesis, this study revealed that children with ADHD have, in contrast to their controls, a general decreased cortical volume and surface area and that these abnormalities are mostly based on differences in younger years. It further enlightened that the temporal lobe is an ADHD typical region, as this region showed a decrease in volume, thickness and surface area in children with ADHD, but not in children with ASD. With a SBM approach, a previously unsuspected insular cortex showed an ADHD specific decrease in cortical thickness and volume.

In contrast to the postulated hypothesis, previous findings of a general increased cortical volume in children with ASD could not be replicated, probably due to a too old cohort group, as disorder specific differences are expected to be most pronounced between ages of 2 and 3 years (Courchesne et al., 2001). Furthermore, this study was not able to find the predicted decreased volume of the corpus callosum in either disorder. This could be explained by the fact that FreeSurfer's algorithm might not be as sensitive and accurate in calculating actual corpus callosum volume. But it is more likely that differences in the corpus callosum are not represented by volume abnormalities, but rather manifested in aberrant connections inside this volume. Therefore, a better approach would be to analyze the properties of this structure by using a DTI approach.

The present study was further capable of defining regions that are unifying the two disorders and differentiating them from each other. This provides future studies with the opportunity of defining target regions that could explain why symptoms of ADHD seem to be more strongly present in ASD than vice-versa and why in some cases, symptoms of ADHD disappear with age (Frodl & Skokauskas, 2012), while the diagnosis of ASD is almost always a lifetime condition (Seltzer et al., 2003).

The analysis of global gray and white matter volume showed that the structural abnormalities found in either disorder are most certainly based on GM abnormalities. Future DTI studies will be needed to determine whether this is the case of if local abnormalities are based on differences in the fiber tracts of white matter regions.

Closer analysis of the results seen in *Supplementary B11* have shown that significant differences were found in the left hemisphere with a much higher frequency than in the right hemisphere. This could be explained by a lateralization effect in either ASD or ADHD and would mean that future studies should look at lateralization and asymmetry between and within either disorder.

Further, the current study enlightened that structural brain studies should not only focus on volume measurements, but also on cortical thickness and surface area. Thus, SBM analyses seem to be a better approach than VBM methods to find brain specific characteristics (Jiao et al., 2010). Altogether, longitudinal studies with well-matched groups of children with ADHD, ASD and TDC, beginning at the age of 2 years, would be the best approach to confirm previous findings. Further, the degree of having either ADHD or ASD should be assessed. Munson et al. (2006) reported that a larger amygdalar volume is predictive for a stronger autistic symptomatic and Hardan et al. (2009) showed that greater cortical thinning is correlated with more sever symptomatology, whereas Barttfeld, Wicker, Cukier, Navarta, Lew and Sigman (2011) reported that an ASD severity increase is associated with an increase in short-range and a decrease of long-range coherence in the EEG signal. Moreover, this study also showed that in terms of neuroanatomical characteristics, cortical folding (Nordahl, Dierker, Mostafavi, Schumann, Rivera, Amaral & Van Essen, 2007), neurotransmitters (Penn, 2006) or cellular information such as cell density or neuron size (Sokol & Edwards-Brown, 2004) would be of great interest. Further, including family history, perinatal complications or genetic variations of affected children (Amico et al., 2011) and molecular genetics as well (Faraone, Perlis, Doyle, Smoller, Goralnick, Holmgren & Sklar, 2005) could provide interesting insights. As reported by Brieber et al. (2007), ADHD and ASD have both a strong genetic component and even show some overlap in the affected genes. A longitudinal sibling study, where the children are diagnosed with either ADHD or ASD or both, might provide enormous insight on the similarities and differences between the two disorders and might account for the fact that brain volume and cortical thickness are highly heritable characteristic (Wallace et al., 2010). Additionally, future studies should investigate differences and similarities between ADHD and ASD by using other promising imaging technics, such as the analysis of functional resting states, network connectivity, DTI and cortical folding. By pooling all the gained information from such methods, we might be capable of developing disorder specific neurological markers and therefore be able to detect ADHD and ASD in children much earlier. This early detection is of high importance, as it might allow to positively influencing the abnormal growth trajectory already in early years. Hopefully, future studies will be able to answer questions such as: Why does ASD almost always persist into adulthood, while ADHD does not? In what way do pure cases of ASD only or ADHD only exist and where on a spectrum between ADHD and ASD do the disorder specific subtypes belong to?

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6. Supplementary Material

A1: *Number of subjects per scanner site from the ABIDE and the ADHD-200 dataset*

	Number of subjects
ABIDE	
California Institute of Technology	38
Carnegie Mellon University	27
Kennedy Krieger Institute	55
Ludwig Maximilians, University Munich	57
NYU Langone Medical Center	184
Olin, Institute of Living, at Hartford Hospital	36
Oregon Health and Science University	28
San Diego State University	36
Social Brain Lab, BCN NIC UMC Groningen and Netherlands Institute for Neurosciences	30
Stanford University, School of Medicine	40
Trinity Centre for Health Sciences	49
University of California, Los Angeles	109
University of Leuven	64
University of Michigan	145
University of Pittsburgh, School of Medicine	57
University of Utah, School of Medicine	101
Yale School of Medicine, Child Study Center	56
ADHD-200	
Bradley Hospital/Brown University	26
Kennedy Krieger Institute	94
NeuroIMAGE sample	73
New York University Child Study Center	263
Oregon Health & Science University	113
Peking University	245
University of Pittsburgh	98
Washington University in St. Louis	61

The data was acquired from the ABIDE and ADHD-200 homepage.

A2: *Diagnosis tool used by the sites included in the final dataset*

	Diagnosis tool
ABIDE	
Kennedy Krieger Institute	ADI-R, ADOS, DICA-IV & DSM-IV-TR
NYU Langone Medical Center	ACDS, DSM-IV-TR, K-SADS-PL & SCID-I/NP
Oregon Health and Science University	ADI-R, ADOS, clinical opinion, K-SADS-E, ADHD-RS-IV, Conners
Trinity Centre for Health Sciences	ADI-R, ADOS, DSM-IV-TR & SRS or SCQ
University of California, Los Angeles	ADI-R & ADOS
University of Leuven	AQ, DSM-IV-TR, SCQ, SRS
University of Michigan	ADI-R, ADOS & SCQ or SCAS
University of Pittsburgh, School of Medicine	ADI-R, ADOS & clinical opinion
University of Utah, School of Medicine	ADI-R, ADOS & DSM-IV-TR
ADHD-200	
Kennedy Krieger Institute	CPRS-R, DICA-IV, ADHD-RS-IV & interview
New York University Child Study Center	CPRS-LV & K-SADS-PL
Oregon Health & Science University	Conners, K-SADS-I, interview
Peking University	K-SADS-PL & ADHD-RS-IV

The data was acquired from the ABIDE and ADHD-200 homepage. Abbreviations represent the following: ***ACDS****: Adult ADHD Clinical Diagnostic Scale (Adler & Spencer, 2004);* ***ADHD-RS-IV****: ADHD Rating Scale-IV (DuPaul, Power, Anastopolous & Reid, 1998) New York: Guilford;* ***ADI-R****: Autism Diagnostic Interview-Revised (Le Couteur, Lord & Rutter, 2003; Lord, Rutter, & Le Couteur, 1994);* ***ADOS****: Autism Diagnostic Observation Schedule (Gotham, Pickles & Lord, 2009; Gotham, Risi, Pickles & Lord , 2007; Lord et al, 2000; Lord, Rutter, DiLavore & Risi, 1999);* ***AQ****: Autism Quotient (Baron-Cohen, Wheelwright, Skinner, Martin & Clubley ,2001);* ***clinical opinion****: a clinical review by an expert, a child psychiatrist or a neuropsychologist;* ***Conners****: Conners Manual, 3rd Edition (Conners, 2008);* ***CPRS-LV****: Conners' Rating Scales-Revised (Conners, 1997);* ***CPRS-R****: The revised Conners' Parent Rating Scale (Conners, Sitarenios, Parker & Epstein, 1998);* ***DICA-IV****: Diagnostic interview for children-IV (Reich, Welner & Herjanic, 1997);* ***DSM-IV-TR****: Diagnostic and statistical manual of mental disorders, Revised (APA, 2000);* ***interview****: a structured parent interview based on DSM-IV (APA, 1994) criteria;****K-SADS-E****: Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version, Fifth Revision (Orvaschel, 1994);* ***K-SADS-I****: The Schedule for Affective Disorders and Schizophrenia for School-Age Children (Puig-Antich & Ryan, 1986);* ***K-SADS-PL****: Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (Kaufman et al., 1997);* ***SCAS****: Structure of anxiety symptoms among children (Spence 1997);* ***SCID-I/NP****: Structured Clinical Interview for DSM-IV Axis I Disorders - Non-Patient Edition (First, Spitzer, Gibbon & Williams, 1995);* ***SCQ****: Social Communication Questionnaire (Rutter, Bailey & Lord, 2003);* ***SRS****: Social Responsiveness Scale (Constantino & Gruber 2005)*

A3: *IQ measurement of the sites included in the final dataset*

	IQ Measurement
ABIDE	
Kennedy Krieger Institute	WISC-IV
NYU Langone Medical Center	WASI
Oregon Health and Science University	WISC-IV
Trinity Centre for Health Sciences	WISC-IV
University of California, Los Angeles	WASI or WISC-IV
University of Leuven	GIT or WAIS-III
University of Michigan	PPVT, RSPM & WASI
University of Pittsburgh, School of Medicine	WASI
University of Utah, School of Medicine	WASI & WASI-III
ADHD-200	
Kennedy Krieger Institute	WISC-IV
New York University Child Study Center	WASI
Oregon Health & Science University	WASI
Peking University	WISCC-R

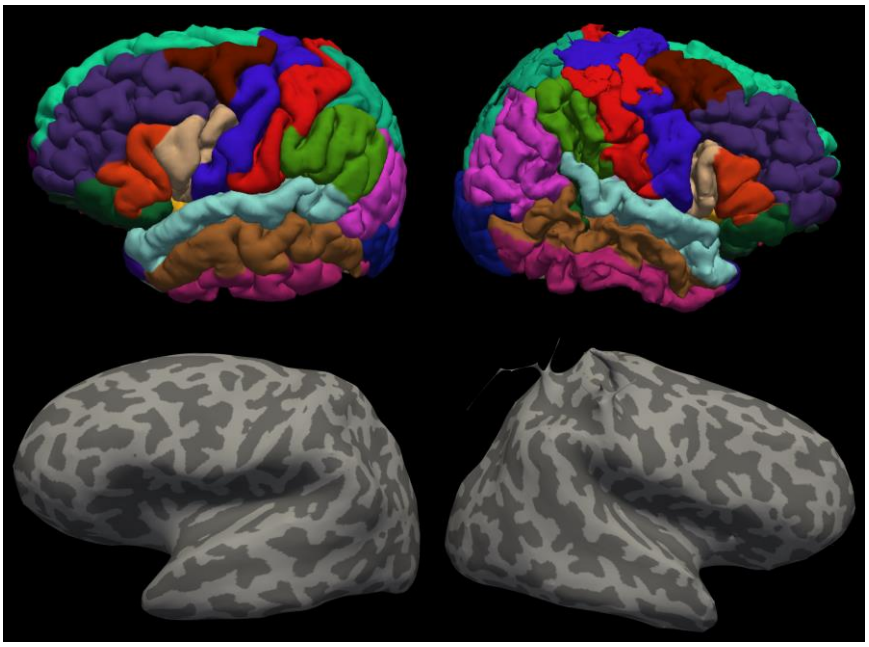
The data was acquired from the ABIDE and ADHD-200 homepage. Abbreviations represent the following: ***GIT 2****: Groninger Intelligence Test 2 (Luteijn & Bartelds, 2004);* ***PPVT****: Peabody picture vocabulary test (3rd ed.) (Dunn & Dunn, 1997);* ***RSPM****: Ravens Standard Progressive Matrices (Raven, 1960);* ***WAIS-III****: Wechsler Adult Intelligence Scale, Third Edition (Wechsler 1997);* ***WASI****: Wechsler Abbreviated Scale of Intelligence manual (Wechsler, 1999);* ***WISCC-R****: Wechsler Intelligence Scale for Chinese Children-Revised (Gong & Cai, 1993);* ***WISC-III****: Wechsler intelligence scale for children, Third Edition (Wechsler 1991);* ***WISC-IV****: Wechsler Intelligence Scale for Children, Fourth Edition (Wechsler 2003)*

A4: *Scan parameters during of the anatomical sMRI scan for each site*

Anatomical Scan	*Voxel size*	*FOV*	*Slices*	*TE [ms]*	*Flip Angle*	*TR [ms]*
ABIDE						
KKI	1.0x1.0x1.0	256x200	200	3.7	8°	shortest (8)
OHSU	1.0x1.0x1.1	256x240	160	3.58	10°	2300
NYU1	1.3x1.0x1.3	256x256	128	3.25	7°	2530
TRIN	1.0x1.1x1.0	256x256	160	3.9	8°	8.5
UCLA	1.0x1.0x1.2	256x240	160	2.84	9°	2300
LEUV	1.0x1.0x1.2	250x250	182	4.6	8°	shortest (9.6)
UM	1.0x1.0x1.2	256x256	40	1.8	15°	250
PITT	1.1x1.1x1.1	269x269	176	3.93	7°	2100
UMS	1.0x1.0x1.2	256x240	160	2.91	9°	2300
ADHD-200						
KKI	1.0x1.0x1.0	256x200	200	3.7	8°	shortest (8)
NYU2	1.3x1.0x1.3	256x256	128	3.25	7°	2530
OHSU	1.0x1.0x1.1	256x240	160	3.58	10°	2300
PEKI part 1	1.0x0.9x0.9	240×240	192	3.67	12°	2000
PEKI part 2	1.3x1.0x1.0	256×256	128	3.37	7°	2530
PEKI part 3	1.0x1.0x1.0	256×256	176	3.92	12°	1770
PEKI part 1	1.0x0.9x0.9	240×240	192	3.67	12°	2000
PEKI part 2	1.3x1.0x1.0	256×256	128	3.37	7°	2530
PEKI part 3	1.0x1.0x1.0	256×256	176	3.92	12°	1770

***KKI**: Kennedy Krieger Institute; **LEUV**: University of Leuven; **NYU1**: NYU Langone Medical Center; **NYU2**: New York University Child Study Center; **OHSU**: Oregon Health and Science University; **PEKI**: Peking University; **PITT**: University of Pittsburgh, School of Medicine; **TRIN**: Trinity Centre for Health Sciences; **UCLA**: University of California, Los Angeles; **UM**: University of Michigan; **USM**: University of Utah, School of Medicine. The data was acquired from the ABIDE and ADHD-200 homepage*

A5: *Examples of sufficient and insufficient results of FreeSurfer's parcellation or segmentation algorithm.*



Top: *Example of a sufficient (left) and insufficient (right) reconstruction of the cortical parcellation of the pial surface according to the Desikan-Killiany Atlas (Desikan et al., 2006).* **Bottom:** *Example of a sufficient (left) and insufficient (right) recreation of an inflated gray matter surface model. Gyri are shown in light and sulci in dark gray.*

A6: *Overview of the number of excluded subjects per criteria*

	ABIDE	*ADHD-200*	*Total*
Total number of subjects in the databases	1112	973	2085
Subject criteria			
Age not in the range between 7 to 18 years	360	116	476
Main diagnosis unknown		26	26
Gender and IQ not known	6	47	53
IQ lower than 85	64	30	94
Comorbidity that interfered with clear group affiliation	16	3	19
Total exclusion	446	222	668
Scanner site criteria			
Less than 5 subjects in group ADHD, ASD or TDC	2	127	129
Total exclusion	2	127	129
Dataset criteria			
No structural MRI scan available	6		6
Doubles in the dataset from New York	35	6	41
Double in dataset of University of Michigan	2		2
Total exclusion	43	6	49
Preprocessing criteria			
FreeSurfer's recon-all algorithm failed	7	4	11
Signal-to-noise ratio smaller than 16	222	145	367
FreeSurfer's parcellation or segmentation created insufficient results	97	19	116
Exclusion of subjects with ADHD-H or PPD-NOS	6	14	20
Less than 5 subjects in group ADHD, ASD or TDC	25	0	25
Total exclusion	357	182	539
Total number of subjects in final dataset	264	436	700

The criteria were applied one after the other. This means that there are probably more subjects with a SNR smaller 16, but they were already excluded because of previous criteria.

A7: *Characteristics of the groups in the final dataset, broken down for each site*

	TDC				*ASD*			
ABIDE	*Subjects [#]*	*Sex [m/f]*	*Age [year]*	*IQ [FSIQ]*	*Subjects [#]*	*Sex [m/f]*	*Age [year]*	*IQ [FSIQ]*
Kennedy Krieger Institute	27	20/7	10.1 (1.2) [8.1-12.8]	115.4 (8)	8	6/2	10.3 (1.3) [8.2-12]	105.5 (14.2)
Oregon Health and Science University	10	10/0	15.3 (1.3) [12.4-16.9]	115 (6.8)	6	6/0	13.4 (1.2) [12.1-14.9]	106.5 (11.7)
NYU Langone Medical Center	19	16/3	12.2 (2.8) [8-17.7]	114.7 (14.3)	20	19/1	11.8 (2.2) [8.8-16.3]	107.8 (12.8)
Trinity Centre for Health Sciences	15	15/0	10.1 (1) [8.2-12]	115.7 (10.7)	8	8/0	11.1 (2) [8-14]	117.2 (11.8)
University of California, Los Angeles	15	12/3	14 (2) [9.4-17.4]	106.9 (8.4)	17	13/4	14.1 (2.4) [9.3-17.8]	108.1 (11.8)
University of Leuven	15	15/0	14.5 (1.6) [12-17.5]	108.1 (13)	11	11/0	14.8 (1.6) [12-17.3]	106 (14.5)
University of Michigan	22	17/5	13.6 (1.9) [11.3-17.8]	105.9 (9.6)	29	26/3	13 (2.5) [8.5-17.5]	105.4 (11.6)
University of Pittsburgh, School of Medicine	7	5/2	16.2 (1.3) [14.3-17.9]	108.4 (7.6)	5	4/1	14 (1.3) [12.6-16.1]	108.1 (11.2)
University of Utah, School of Medicine	11	11/0	13.9 (2.6) [9.9-17.1]	114.9 (14)	11	11/0	15.9 (1.8) [11.4-17.6]	103.7 (11)
	TDC				ADHD			
ADHD-200	*Subjects [#]*	*Sex [m/f]*	*Age [year]*	*IQ [FSIQ]*	*Subjects [#]*	*Sex [m/f]*	*Age [year]*	*IQ [FSIQ]*
Kennedy Krieger Institute	60	35/25	10.4 (1.3) [8-12.9]	111.7 (10.2)	20	12/8	10.1 (1.5) [8.1-13]	103.9 (14.5)
New York University Child Study Center	48	21/27	12.7 (2.7) [8.2-17.9]	112.9 (12.7)	59	44/15	11.6 (2.7) [8.1-17.6]	109.8 (12)
Oregon Health and Science University	48	23/25	9.5 (1.2) [8.1-12.5]	115.1 (12.6)	27	20/7	9.4 (1.1) [8-11.8]	111.5 (12)
Peking University	115	59/56	11.3 (1.9) [8.1-15.2]	118.3 (12.4)	67	58/9	12 (2.1) [8.3-17.3]	106.8 (11.9)

The column age and IQ represent the mean of the sample with the standard deviation written inside parentheses. For the column age, the range of years is written inside brackets.

B1: *Comparison of age, IQ and gender between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Age	[years (SD)]	WD	BD	[years (SD)]	WD	BD	[years (SD)]	WD	BD
ADHD	11.22 (2.39)	1.01 0.313[a]	**-6.54 2.8E-10** *0.361*	10.03 (1.32)	-1.46 0.145	**-4.27 3.1E-5** *0.303*	14.51 (1.36)	0.15 0.883	**-2.78 0.006** *0.264*
TDC_{ADHD}	11 (2.1)			10.25 (1.37)			14.47 (1.21)		
ASD	13.17 (2.59)	1.15 0.253		10.93 (1.3)	1.31 0.193		15.29 (1.48)	-0.1 0.921	
TDC_{ASD}	12.78 (2.74)			10.61 (1.41)			15.32 (1.39)		
IQ	[FSIQ (SD)]	WD	BD	[FSIQ (SD)]	WD	BD	[FSIQ (SD)]	WD	BD
ADHD	108.22 (12.54)	**-5.89 7.8E-9** *0.270*	0.74 0.463	108.53 (12.93)	**-5.35 1.6E-7** *0.276*	0.67 0.502	107.37 (11.45)	**-2.02 0.046** *0.206*	0.12 0.904
TDC_{ADHD}	115.34 (12.36)			115.99 (12.32)			112.31 (12.24)		
ASD	107.1 (12.72)	**-3.07 0.002** *0.189*		107.12 (13.17)	**-3.16 0.002** *0.267*		107.08 (12.4)	-1.05 0.296	
TDC_{ASD}	111.76 (11.46)			113.84 (11.19)			109.32 (11.37)		
Gender	[males (%)]	WD	BD	[males (%)]	WD	BD	[males (%)]	WD	BD
ADHD	134 (77%)	**31.33 2.2E-8**	**8.11 0.004**	91 (72%)	**15.25 9.4E-5**	**4.21 0.040**	43 (93%)	**18.59 1.6E-5**	010 0.753
TDC_{ADHD}	138 (51%)			112 (50%)			26 (54%)		
ASD	104 (90%)	1.27 0.260		48 (86%)	0.06 0.812		56 (95%)	2 0.158	
TDC_{ASD}	121 (86%)			64 (84%)			57 (88%)		

WD = *Comparison within each disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD}.;* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value for age and IQ and the χ^2-value for gender, the second value is the 2-tailed p-value and the third value is the effect-size r (in cursive). Bold values indicate p <0.05;* ***a*** = *Leven's Test for Equality of Variance showed p < 0.05, equal variances therefore could not be assumed;* ***SD*** = *standard deviation.*

B2: *Comparison of global cortical GM and WM measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
TGMV	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	723.692 (4.392)	**-2.02 0.044** *0.076*	**-3.09 0.002** *0.116*	728.174 (5.182)	**-2.54 0.011** *0.115*	**-2.54 0.011** *0.115*	714.501 (8.251)	0.28 0.781	-1.24 0.218
TDC_{ADHD}	735.407 (3.683)			745.145 (3.978)			711.11 (8.659)		
ASD	745.415 (5.525)	0.03 0.973		751.88 (7.851)	-0.11 0.916		728.046 (7.295)	-0.05 0.961	
TDC_{ASD}	745.168 (4.891)			752.954 (6.637)			728.536 (6.862)		
TWMV	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	426.74 (3.567)	-1.37 0.171	-1.38 0.167	414.401 (4.04)	-1.63 0.103	-1.18 0.239	453.392 (7.387)	-0.13 0.896	-0.62 0.534
TDC_{ADHD}	433.206 (2.992)			422.896 (3.101)			454.822 (7.752)		
ASD	434.641 (4.488)	-1.17 0.244		422.96 (6.121)	-0.47 0.636		459.498 (6.531)	-1.28 0.203	
TDC_{ASD}	441.425 (3.973)			426.7 (5.174)			470.801 (6.143)		
MCT	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.697 (0.0076)	0.89 0.374	**-2.96 0.003** *0.111*	2.741 (0.0091)	0.87 0.386	-1.56 0.119	2.598 (0.0142)	0.31 0.755	**-2.62 0.01** *0.175*
TDC_{ADHD}	2.688 (0.0064)			2.73 (0.007)			2.592 (0.0149)		
ASD	2.733 (0.0096)	1.24 0.216		2.766 (0.0138)	0.76 0.449		2.648 (0.0126)	0.84 0.404	
TDC_{ASD}	2.718 (0.0085)			2.753 (0.0117)			2.633 (0.0118)		
TCSA	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	1805.53 (11.6)	**-2.7 0.007** *0.102*	-0.92 0.358	1789.49 (13.55)	**-3.3 0.001** *0.149*	-1.06 0.292	1844.84 (22.36)	0.20 0.840	0.40 0.686
TDC_{ADHD}	1846.94 (9.73)			1847.12 (10.4)			1838.16 (23.46)		
ASD	1822.61 (14.59)	-0.78 0.433		1815.21 (20.53)	-0.79 0.428		1832.85 (19.77)	-0.46 0.644	
TDC_{ASD}	1837.42 (12.91)			1836.23 (17.36)			1845.26 (18.59)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* **TGMV***: Total gray matter volume;* **TWMV***: Total white matter volume;* **MCT***: Mean cortical thickness* **TCSA***: Total cortical surface area;* **SE** = *standard error.*

B3: *Comparison of cerebellar GM and cerebellar WM between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Cerebellar GM	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	108.8	0.14	-1.92	108.7	0.19	-1.35	108.7	-0.2	-1.46
	(0.88)	0.892	0.055	(0.98)	0.849	0.179	(1.91)	0.842	0.146
TDC_{ADHD}	108.7			108.4			109.3		
	(0.74)			(0.75)			(2.00)		
ASD	111.6	-0.14		111.0	-0.01		112.4	-0.37	
	(1.11)	0.887		(1.48)	0.993		(1.69)	0.711	
TDC_{ASD}	111.8			111.0			113.3		
	(0.98)			(1.25)			(1.59)		
Cerebellar WM	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	28.46	-0.32	-1.85	28.07	-0.21	-1.43	29.04	-0.01	-1.06
	(0.28)	0.746	0.065	(0.31)	0.833	0.153	(0.59)	0.993	0.292
TDC_{ADHD}	28.48			28.16			29.05		
	(0.24)			(0.24)			(0.62)		
ASD	29.19	-0.65		28.89	0.00		29.86	-1.00	
	(0.35)	0.519		(0.48)	0.996		(0.52)	0.321	
TDC_{ASD}	29.48			28.89			30.56		
	(0.31)			(0.40)			(0.49)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* **SE** = *standard error.*

B4: *Comparison of ratio between cortical GM to cortical WM between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Cortex	Ratio GM to WM (SE)	WD	BD	Ratio GM to WM (SE)	WD	BD	Ratio GM to WM (SE)	WD	BD
ADHD	1.457	-0.18	-1.27	1.508	-0.65	-0.98	1.347	0.64	-0.25
	(0.0079)	0.854	0.203	(0.0097)	0.518	0.330	(0.0137)	0.526	0.804
TDC_{ADHD}	1.459			1.516			1.334		
	(0.0066)			(0.0074)			(0.0144)		
ASD	1.473	1.70		1.525	0.43		1.351	**2.16**	
	(0.0099)	0.089		(0.0146)	0.665		(0.0121)	**0.032**	
								0.145	
TDC_{ASD}	1.451			1.517			1.316		
	(0.0088)			(0.0124)			(0.0114)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; Column WD and BD show the 2-tailed p-value. Bold values indicate p < 0.05;* **SE** = *standard error.*

B5a: *Comparison of Frontal Lobe measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.823 (0.006)	**2.29 0.022** *0.086*	**4.84 1.6E-6** *0.180*	2.855 (0.007)	**2.79 0.005** *0.126*	**2.69 0.007** *0.122*	2.753 (0.012)	-0.42 0.675	**3.51 0.001** *0.232*
TDC_{ADHD}	2.805 (0.005)			2.831 (0.005)			2.761 (0.013)		
ASD	2.777 (0.008)	0.76 0.446		2.822 (0.01)	1.01 0.313		2.697 (0.011)	0.29 0.77	
TDC_{ASD}	2.77 (0.007)			2.809 (0.009)			2.692 (0.01)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	638.33 (1.454)	0.05 0.961	**-2.72 0.007** *0.102*	636.38 (1.667)	0.03 0.973	**-2.01 0.045** *0.091*	642.84 (3.003)	0.21 0.833	-1.64 0.102
TDC_{ADHD}	638.24 (1.217)			636.31 (1.275)			641.9 (3.151)		
ASD	644.65 (1.823)	-0.14 0.891		642.38 (2.507)	0.20 0.840		649.39 (2.655)	-0.44 0.660	
TDC_{ASD}	644.97 (1.614)			641.72 (2.119)			650.97 (2.497)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	210.12 (0.676)	1.47 0.142	0.71 0.478	212.46 (0.779)	1.52 0.130	-0.13 0.896	205.27 (1.377)	0.4 0.686	1.11 0.267
TDC_{ADHD}	208.81 (0.564)			210.94 (0.594)			204.44 (1.448)		
ASD	209.35 (0.848)	0.55 0.584		212.64 (1.173)	0.9 0.367		203.23 (1.218)	-0.03 0.977	
TDC_{ASD}	208.75 (0.751)			211.28 (0.992)			203.28 (1.146)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** = *standard error.*

B5b: *Comparison of Temporal Lobe measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.994 (0.007)	**-3.87 1.2E-4** *0.145*	-1.02 0.309	3.031 (0.008)	**-3.82 1.5E-4** *0.172*	-0.17 0.869	2.917 (0.014)	-0.62 0.535	-0.76 0.448
TDC_{ADHD}	3.029 (0.006)			3.069 (0.006)			2.929 (0.014)		
ASD	3.005 (0.009)	**-1.97 0.049** *0.074*		3.033 (0.012)	-1.72 0.087		2.93 (0.012)	-1.25 0.214	
TDC_{ASD}	3.027 (0.008)			3.059 (0.01)			2.951 (0.011)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	353.55 (0.965)	-1.38 0.168	-0.63 0.529	353.25 (1.104)	**-2.05 0.041** *0.093*	-0.18 0.861	354.45 (1.988)	0.86 0.392	-0.23 0.822
TDC_{ADHD}	355.32 (0.809)			356.18 (0.845)			351.93 (2.087)		
ASD	354.52 (1.211)	1.14 0.255		353.6 (1.661)	0.43 0.671		355.04 (1.758)	1.01 0.311	
TDC_{ASD}	352.74 (1.072)			352.69 (1.404)			352.63 (1.654)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	127.73 (0.469)	**-2.87 0.004** *0.108*	0.13 0.898	128.98 (0.549)	**-3.57 4.0E-4** *0.161*	0.20 0.840	125.33 (0.908)	0.95 0.343	0.67 0.504
TDC_{ADHD}	129.51 (0.391)			131.5 (0.418)			124.06 (0.955)		
ASD	127.64 (0.588)	0.38 0.703		128.78 (0.826)	-0.18 0.860		124.52 (0.803)	0.67 0.504	
TDC_{ASD}	127.35 (0.52)			128.97 (0.699)			123.79 (0.756)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** = *standard error.*

B5c: *Comparison of Parietal Lobe measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.537	0.9	-1.14	2.577	0.31	-1.02	2.448	1.24	-0.54
	(0.004)	0.368	0.253	(0.005)	0.754	0.309	(0.008)	0.217	0.588
TDC_{ADHD}	2.533			2.576			2.434		
	(0.003)			(0.004)			(0.008)		
ASD	2.545	-0.04		2.586	0.48		2.454	-0.46	
	(0.005)	0.972		(0.007)	0.631		(0.007)	0.643	
TDC_{ASD}	2.545			2.582			2.459		
	(0.004)			(0.006)			(0.007)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	489.46	**2.37**	**2.48**	488.59	**2.05**	0.42	491.78	0.50	**3.33**
	(1.218)	**0.018**	**0.014**	(1.442)	**0.041**	0.674	(2.263)	0.619	**0.001**
		0.089	*0.093*		*0.093*				*0.220*
TDC_{ADHD}	485.63			484.78			490.12		
	(1.02)			(1.103)			(2.375)		
ASD	484.64	1.35		487.51	0.95		481.78	1.24	
	(1.528)	0.178		(2.169)	0.341		(2.001)	0.216	
TDC_{ASD}	481.97			484.84			478.42		
	(1.352)			(1.834)			(1.882)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	141.47	1.73	1.38	144.24	1.37	0.21	135.7	0.95	**2.14**
	(0.478)	0.084	0.167	(0.569)	0.170	0.832	(0.894)	0.341	**0.034**
									0.144
TDC_{ADHD}	140.38			143.23			134.44		
	(0.399)			(0.433)			(0.939)		
ASD	140.41	0.51		144.02	0.51		133.16	0.45	
	(0.599)	0.609		(0.856)	0.613		(0.79)	0.655	
TDC_{ASD}	140.01			143.46			132.68		
	(0.531)			(0.724)			(0.744)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** = *standard error.*

B5d: *Comparison of Occipital Lobe measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.184 (0.007)	**2.16 0.031** *0.081*	**-3.07 0.002** *0.115*	2.229 (0.008)	1.8 0.073	-1.84 0.066	2.085 (0.015)	1.21 0.226	**-2.06 0.040** *0.139*
TDC_{ADHD}	2.164 (0.006)			2.211 (0.006)			2.058 (0.016)		
ASD	2.219 (0.009)	0.45 0.656		2.255 (0.012)	-0.02 0.987		2.128 (0.014)	0.57 0.567	
TDC_{ASD}	2.214 (0.008)			2.255 (0.01)			2.117 (0.013)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	225.94 (1.106)	-1.20 0.232	1.48 0.140	225 (1.273)	-0.66 0.507	**1.98 0.048** *0.090*	227.22 (2.222)	-1.10 0.272	-0.42 0.674
TDC_{ADHD}	227.69 (0.926)			226.09 (0.974)			230.83 (2.332)		
ASD	223.33 (1.387)	**-2.32 0.021** *0.087*		220.49 (1.914)	-1.58 0.115		228.46 (1.965)	-1.71 0.089	
TDC_{ASD}	227.5 (1.228)			224.4 (1.618)			233.01 (1.848)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	53.84 (0.346)	0.42 0.672	-0.53 0.598	54.91 (0.392)	0.41 0.680	0.85 0.394	51.45 (0.7)	0.60 0.550	-1.53 0.126
TDC_{ADHD}	53.64 (0.288)			54.71 (0.299)			50.83 (0.736)		
ASD	54.13 (0.433)	-1.29 0.199		54.31 (0.59)	-1.30 0.194		52.87 (0.619)	-0.54 0.589	
TDC_{ASD}	54.85 (0.384)			55.3 (0.499)			53.33 (0.582)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** = *standard error.*

B5e: *Comparison of Cingulate cortex measurements between ADHD, ASD and TDC*

	All ages			**Age 8 to 12**			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	2.725 (0.011)	**-2.95 0.003** *0.111*	**-4.40 1.2E-5** *0.164*	2.759 (0.013)	**-2.72 0.007** *0.123*	-1.86 0.063	2.641 (0.019)	-1.21 0.229	**-4.68 5.0E-6** *0.303*
TDC_{ADHD}	2.766 (0.009)			2.803 (0.01)			2.674 (0.02)		
ASD	2.8 (0.013)	-0.53 0.597		2.801 (0.019)	-1.21 0.227		2.76 (0.017)	0.29 0.769	
TDC_{ASD}	2.809 (0.012)			2.831 (0.016)			2.753 (0.016)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	79.17 (0.394)	-0.41 0.681	0.75 0.453	79.29 (0.454)	0.6 0.551	**1.99 0.047** *0.090*	79.02 (0.79)	-1.05 0.294	-1.06 0.292
TDC_{ADHD}	79.39 (0.33)			78.94 (0.347)			80.24 (0.829)		
ASD	78.7 (0.494)	-1.01 0.314		77.68 (0.683)	-1.52 0.128		80.12 (0.698)	0.04 0.972	
TDC_{ASD}	79.35 (0.437)			79.02 (0.577)			80.09 (0.657)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	24.37 (0.158)	**-3.08 0.002** *0.116*	-0.28 0.780	24.71 (0.187)	**-2.35 0.019** *0.106*	1.73 0.083	23.61 (0.289)	-1.34 0.182	**-2.30 0.023** *0.154*
TDC_{ADHD}	25.01 (0.132)			25.27 (0.143)			24.18 (0.304)		
ASD	24.44 (0.198)	-1.69 0.091		24.12 (0.282)	**-2.67 0.008** *0.121*		24.49 (0.256)	0.23 0.818	
TDC_{ASD}	24.87 (0.175)			25.09 (0.238)			24.41 (0.241)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** = *standard error.*

B5f: *Comparison of Insular cortex measurements between ADHD, ASD and TDC*

	All ages			Age 8 to 12			**Age 13 to 18**		
Thickness	mm (SE)	WD	BD	mm (SE)	WD	BD	mm (SE)	WD	BD
ADHD	3.162 (0.013)	**-3.52 4.6E-4** *0.132*	**-2.77 0.006** *0.104*	3.212 (0.016)	**-2.68 0.008** *0.121*	-1.03 0.305	3.044 (0.023)	**-2.43 0.016** *0.163*	**-3.15 0.002** *0.209*
TDC_{ADHD}	3.224 (0.011)			3.269 (0.013)			3.127 (0.024)		
ASD	3.221 (0.017)	-0.05 0.961		3.243 (0.025)	-0.55 0.585		3.142 (0.021)	0.30 0.766	
TDC_{ASD}	3.222 (0.015)			3.26 (0.021)			3.134 (0.019)		
Area	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD	cm^2 (SE)	WD	BD
ADHD	44.33 (0.278)	-0.53 0.596	-1.38 0.167	43.99 (0.329)	-0.52 0.600	-1.47 0.144	44.95 (0.531)	-0.36 0.722	-0.71 0.477
TDC_{ADHD}	44.52 (0.232)			44.21 (0.252)			45.23 (0.557)		
ASD	44.94 (0.348)	1.52 0.130		44.85 (0.495)	1.58 0.114		45.45 (0.469)	0.49 0.623	
TDC_{ASD}	44.25 (0.308)			43.83 (0.418)			45.14 (0.441)		
Volume	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	14.38 (0.099)	**-2.92 0.004** *0.110*	-0.98 0.325	14.46 (0.122)	**-2.45 0.015** *0.111*	-0.69 0.491	14.21 (0.172)	-1.38 0.168	-0.72 0.470
TDC_{ADHD}	14.77 (0.082)			14.84 (0.093)			14.56 (0.18)		
ASD	14.54 (0.124)	0.78 0.433		14.61 (0.183)	0.55 0.583		14.38 (0.152)	0.47 0.638	
TDC_{ASD}	14.42 (0.11)			14.48 (0.155)			14.28 (0.143)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* ***SE*** *= standard error.*

B6: *Comparison of corpus callosum (CC) measurements between ADHD, ASD and TDC*

	All ages			Age 8 to 12			Age 13 to 18		
CC Total	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	3.08	1.60	0.65	3.002	1.79	-0.23	3.254	-0.06	0.81
	(0.029)	0.111	0.515	(0.034)	0.074	0.820	(0.056)	0.955	0.419
TDC_{ADHD}	3.019			2.924			3.258		
	(0.024)			(0.026)			(0.059)		
ASD	3.05	0.90		3.016	1.47		3.194	0.07	
	(0.036)	0.369		(0.051)	0.142		(0.049)	0.946	
TDC_{ASD}	3.008			2.919			3.189		
	(0.032)			(0.043)			(0.047)		
CC Anterior	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	0.827	0.00	-1.12	0.812	0.19	-1.03	0.866	-0.43	-0.52
	(0.011)	0.997	0.263	(0.013)	0.849	0.303	(0.021)	0.670	0.605
TDC_{ADHD}	0.828			0.808	0.19		0.879		
	(0.009)			(0.01)	0.849		(0.022)		
ASD	0.847	1.28		0.836	0.19		0.88	1.26	
	(0.014)	0.200		(0.02)	0.849		(0.019)	0.208	
TDC_{ASD}	0.824			0.814			0.848		
	(0.012)			(0.017)			(0.018)		
CC Central	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	0.466	**1.98**	1.88	0.458	**2.28**	1.12	0.482	-0.08	0.98
	(0.007)	**0.048** *0.075*	0.060	(0.008)	**0.023** *0.103*	0.264	(0.015)	0.938	0.326
TDC_{ADHD}	0.448			0.435			0.484		
	(0.006)			(0.006)			(0.015)		
ASD	0.445	-0.34		0.442	0.61		0.464	-1.05	
	(0.009)	0.735		(0.012)	0.544		(0.013)	0.294	
TDC_{ASD}	0.449			0.433			0.482		
	(0.008)			(0.01)			(0.012)		
CC Mid-Anterior	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	0.506	**2.59**	1.42	0.497	**2.86**	-0.12	0.53	-0.14	1.91
	(0.009)	**0.010** *0.097*	0.156	(0.011)	**0.004** *0.129*	0.908	(0.017)	0.888	0.058
TDC_{ADHD}	0.476			0.457			0.533		
	(0.008)			(0.008)			(0.017)		
ASD	0.486	0.59		0.499	1.33		0.488	-0.42	
	(0.011)	0.558		(0.016)	0.184		(0.015)	0.673	
TDC_{ASD}	0.477			0.471			0.496		
	(0.01)			(0.014)			(0.014)		
CC Mid-Posterior	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	0.418	0.02	-0.43	0.405	0.19	-0.73	0.444	-0.12	-0.34
	(0.006)	0.986	0.664	(0.007)	0.846	0.464	(0.011)	0.906	0.736
TDC_{ADHD}	0.418			0.404			0.446		
	(0.005)			(0.005)			(0.011)		
ASD	0.422	-0.01		0.414	1.30		0.449	-1.2	
	(0.007)	0.995		(0.01)	0.194		(0.01)	0.233	
TDC_{ASD}	0.422			0.397			0.465		
	(0.006)			(0.008)			(0.009)		
CC Posterior	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	0.862	0.97	0.83	0.831	0.77	0.29	0.932	0.54	0.72
	(0.009)	0.335	0.405	(0.01)	0.440	0.771	(0.02)	0.589	0.473
TDC_{ADHD}	0.85			0.821			0.916		
	(0.008)			(0.008)			(0.021)		
ASD	0.85	0.97		0.826	1.09		0.913	0.63	
	(0.012)	0.332		(0.015)	0.277		(0.017)	0.531	
TDC_{ASD}	0.835			0.804			0.898		
	(0.01)			(0.013)			(0.016)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* **SE** = *Standard error.*

B7: *Comparison of subcortical ROI measurements between ADHD, ASD and TDC*

	All ages			Age 8 to 12			Age 13 to 18		
Accumbens Area	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	1.44 (16.069)	-0.18 0.859	**-4.81 1.8E-6** *0.179*	1.463 (17.711)	0.59 0.555	**-4.21 3.0E-5** *0.189*	1.389 (34.898)	-0.90 0.371	**-2.92 0.004** *0.194*
TDC_{ADHD}	1.444 (13.4)			1.45 (13.492)			1.435 (36.686)		
ASD	1.564 (20.139)	1.39 0.164		1.597 (26.656)	**2.65 0.008** *0.120*		1.525 (30.856)	-0.41 0.685	
TDC_{ASD}	1.528 (17.836)			1.506 (22.558)			1.542 (29.041)		
Amygdala	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	3.24 (0.029)	-1.41 0.159	**-2.70 0.007** *0.102*	3.184 (0.032)	-1.67 0.096	-1.11 0.268	3.35 (0.061)	0.30 0.761	**-2.56 0.011** *0.171*
TDC_{ADHD}	3.293 (0.024)			3.252 (0.024)			3.323 (0.064)		
ASD	3.363 (0.036)	-0.08 0.934		3.247 (0.048)	-0.2 0.844		3.557 (0.054)	-0.14 0.891	
TDC_{ASD}	3.367 (0.032)			3.259 (0.041)			3.567 (0.051)		
Caudate	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	8.282 (0.067)	-0.50 0.616	**-2.52 0.012** *0.095*	8.26 (0.081)	-0.83 0.408	**-2.89 0.004** *0.131*	8.346 (0.122)	0.18 0.859	-0.31 0.754
TDC_{ADHD}	8.326 (0.056)			8.346 (0.062)			8.313 (0.128)		
ASD	8.552 (0.084)	1.50 0.133		8.681 (0.122)	1.66 0.097		8.396 (0.108)	0.54 0.592	
TDC_{ASD}	8.389 (0.074)			8.42 (0.103)			8.318 (0.102)		
Pallidum	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	3.844 (0.034)	-0.76 0.447	1.58 0.115	3.848 (0.04)	-0.11 0.912	0.37 0.708	3.814 (0.063)	**-2.44 0.016** *0.163*	1.61 0.110
TDC_{ADHD}	3.878 (0.028)			3.854 (0.031)			4.041 (0.066)		
ASD	3.757 (0.043)	-0.66 0.509		3.821 (0.061)	-0.47 0.639		3.679 (0.056)	-0.40 0.689	
TDC_{ASD}	3.794 (0.038)			3.858 (0.052)			3.709 (0.053)		
Putamen	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	13.104 (0.098)	-0.91 0.364	-1.29 0.199	13.057 (0.115)	-0.55 0.579	**-2.54 0.012** *0.115*	13.221 (0.188)	-1.26 0.209	0.60 0.546
TDC_{ADHD}	13.221 (0.081)			13.139 (0.088)			13.57 (0.197)		
ASD	13.305 (0.122)	1.05 0.293		13.58 (0.173)	1.94 0.053		13.071 (0.166)	-0.28 0.78	
TDC_{ASD}	13.139 (0.108)			13.148 (0.146)			13.133 (0.156)		

Hippocampus	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	8.215 (0.056)	0.19 0.853	-1.78 0.075	8.107 (0.063)	0.42 0.674	-0.84 0.400	8.378 (0.117)	-0.68 0.494	**-2.20 0.029** *0.148*
TDC_{ADHD}	8.201 (0.047)			8.073 (0.048)			8.496 (0.123)		
ASD	8.375 (0.07)	0.13 0.896		8.202 (0.095)	0.50 0.618		8.721 (0.104)	-0.39 0.699	
TDC_{ASD}	8.363 (0.062)			8.141 (0.08)			8.775 (0.098)		
Thalamus Proper	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD	cm^3 (SE)	WD	BD
ADHD	15.317 (0.084)	-0.10 0.917	**2.19 0.029** *0.082*	15.114 (0.095)	0.55 0.585	1.62 0.106	15.676 (0.172)	-1.73 0.086	0.89 0.377
TDC_{ADHD}	15.328 (0.07)			15.048 (0.072)			16.113 (0.18)		
ASD	15.024 (0.105)	**-2.04 0.042** *0.077*		14.84 (0.142)	-1.29 0.199		15.473 (0.152)	-1.77 0.079	
TDC_{ASD}	15.3 (0.093)			15.076 (0.12)			15.836 (0.143)		

WD = *Comparison within disorder: ADHD to TDC_{ADHD} and ASD to TDC_{ASD};* **BD** = *Comparison between ADHD and ASD; In column WD and BD, the first value is the t-value, the second value is the 2-tailed p-value and the third value (if stated) is the effect-size r (in cursive). Bold values indicate p <0.05;* **SE** = *Standard error.*

B8a: *F-contrast comparing the cortical thickness between all four groups*

Left Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages							**A: All ages**						
rostralmiddlefrontal	113.83	0.0001	5.87E-16	-23.8	53.4	-4.4	rostralmiddlefrontal	135.13	0.0001	1.03E-14	19.9	60.2	-11
lateraloccipital	77.09	0.0001	1.71E-09	-15.7	-102.7	-0.5	lateraloccipital	80.49	0.0001	4.72E-08	14.2	-101.6	-1.4
Insula	61.65	0.0001	3.68E-07	-33.9	-6.9	14.3	insula	13.21	0.0008	1.62E-07	33	-18.7	20.3
postcentral	17.48	0.0001	2.59E-06	-35.7	-23	43.1	inferiortemporal	8.71	0.0333	7.13E-04	43.7	-1.3	-34
superiortemporal	8.10	0.0476	6.52E-05	-48.8	-11.3	-10.7							
middletemporal	10.59	0.0090	5.47E-04	-56	-44.8	-10.9							
B: Age 8 to 12							**B: Age 8 to 12**						
rostralmiddlefrontal	59.87	0.0001	1.14E-08	-38	38.1	6.9	lingual	33.22	0.0001	1.08E-06	12.8	-61.1	3
lingual	42.75	0.0001	7.10E-07	-11.8	-64.1	1.7	parahippocampal	8.71	0.0333	2.32E-06	24.7	-28.5	-21.6
Insula	17.22	0.0001	2.90E-06	-33.3	-7.7	15.7	rostralmiddlefrontal	56.80	0.0001	3.09E-06	28.9	26.9	41.3
unknown	8.81	0.0297	1.48E-05	-34.2	-0.2	-21	middletemporal	9.49	0.0193	1.17E-03	51.3	-6.6	-27.2
superiorfrontal	16.42	0.0001	1.80E-05	-22.4	18.1	40.3							
caudalmiddlefrontal	8.53	0.0354	7.59E-05	-34.1	14.4	31							
fusiform	10.01	0.0137	7.98E-04	-29.4	-68.5	-14.4							
C: Age 13 to 18							**C: Age 13 to 18**						
rostralmiddlefrontal	70.84	0.0001	7.33E-11	-24.8	53.3	-4.3	rostralmiddlefrontal	82.90	0.0001	2.12E-11	19.9	60.2	-11
isthmuscingulate	13.00	0.0016	4.51E-08	-6.5	-50.7	18.3	insula	15.07	0.0003	1.68E-07	34.1	-13.6	12.8
lateraloccipital	28.73	0.0001	3.78E-05	-16.5	-102.1	-5.2	lateraloccipital	12.11	0.0023	3.94E-07	17.8	-101.5	-2
postcentral	10.28	0.0112	1.69E-04	-36	-23.8	43.9	fusiform	10.42	0.0088	1.69E-06	30	-65.8	-14.6
							precentral	13.39	0.0008	5.43E-05	31.8	-17.4	67
							lingual	15.71	0.0003	1.58E-04	20.8	-49.2	5.5

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* $\mathbf{MNI_{X,Y,Z}}$ = *MNI-coordinate of MPC.*

B8b: *T-contrast comparing the cortical thickness of ADHD to TDC_{ADHD}*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
rostralmiddlefrontal	pos	11.97	0.0033	3.27E-07	-38.5	37.4	7.1	lingual	pos	15.94	0.0003	2.34E-07	14.7	-60.9	2.2
parsopercularis	neg	10.06	0.0132	1.14E-06	-45.9	5.1	4.9	parahippocampal	neg	8.51	0.0372	3.15E-06	20.5	-29.2	-17.8
lingual	pos	19.39	0.0001	1.33E-06	-4.8	-95.2	-11	inferiortemporal	neg	14.98	0.0003	2.52E-04	43.7	-1.3	-34
medialorbitofrontal	pos	32.19	0.0001	1.60E-06	-8.3	51.7	-20.2	superiorfrontal	pos	25.40	0.0001	4.38E-04	12.3	63.1	16.9
Insula	neg	50.68	0.0001	1.73E-05	-30.7	18.3	1.6								
B: Age 8 to 12								**B: Age 8 to 12**							
rostralmiddlefrontal	pos	14.20	0.0007	1.33E-08	-39.5	37.7	6.2	parahippocampal	neg	9.78	0.0144	8.00E-07	24.7	-28.5	-21.6
parsopercularis	neg	9.24	0.0231	2.64E-07	-47.5	7.8	2	lingual	pos	8.72	0.0332	1.86E-06	13.7	-60.7	2.6
lateralorbitofrontal	pos	22.68	0.0001	6.47E-07	-8.9	51.7	-20.6	superiortemporal	neg	11.76	0.0034	3.12E-05	50.3	10.7	-13.4
lingual	pos	10.87	0.0074	4.57E-06	-5.5	-94.9	-12.7	superiorfrontal	pos	25.63	0.0001	1.35E-04	8.2	61.5	22.4
temporalpole	neg	28.87	0.0001	1.28E-04	-26.4	9.6	-35.9	inferiortemporal	neg	12.36	0.0020	8.02E-04	47.5	-7.9	-32.4
rostralmiddlefrontal	pos	26.40	0.0001	6.38E-04	-21.8	51	22.1								
fusiform	neg	11.20	0.0060	8.85E-04	-31.6	-34.1	-23.8								
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								no cluster found							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ADHD > TDC_{ADHD}, neg = ADHD < TDC_{ADHD};* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B8c: *T-contrast comparing the cortical thickness of ASD to TDC_{ASD}*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
middletemporal	neg	11.59	0.0051	1.37E-03	-56.2	-23	-19.7	no cluster found							
B: Age 8 to 12								**B: Age 8 to 12**							
no cluster found								no cluster found							
C: Age 13 to 18								**C: Age 13 to 18**							
superiorparietal	neg	11.18	0.0060	7.26E-05	-18	-87	32.6	lateraloccipital	pos	8.14	0.0477	3.22E-03	25.8	-90.1	2.5

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD > TDC_{ASD}, neg = ASD < TDC_{ASD};* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B8d: *T-contrast comparing the cortical thickness of ASD to ADHD*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
rostralmiddlefrontal	neg	89.57	0.0001	4.09E-11	-21.8	53.9	-4.4	rostralmiddlefrontal	neg	120.03	0.0001	2.36E-12	19.6	60.6	-11.4
posteriorcingulate	pos	23.01	0.0001	8.28E-07	-6	-34	33.2	insula	pos	8.45	0.0387	2.62E-06	33.5	-18.1	20.9
lateraloccipital	pos	19.08	0.0001	1.58E-06	-14.2	-103.2	-0.8	lateraloccipital	pos	8.23	0.0448	2.92E-06	16.4	-102.1	-0.9
Insula	pos	17.86	0.0001	4.32E-06	-33.7	-7.7	13.6	precentral	pos	16.55	0.0001	3.58E-05	35.3	-20.9	60.9
postcentral	pos	18.00	0.0001	3.67E-05	-24.2	-32.2	70.9	precuneus	pos	24.64	0.0001	3.84E-05	13.9	-42.6	33.5
B: Age 8 to 12								**B: Age 8 to 12**							
postcentral	pos	11.99	0.0033	9.77E-05	-24.5	-31.3	71.1	rostralmiddlefrontal	neg	62.13	0.0001	2.48E-06	30.2	26.5	43.2
rostralmiddlefrontal	neg	17.92	0.0001	1.60E-04	-20.1	55.1	-5								
lateraloccipital	pos	8.74	0.0314	6.71E-04	-18.3	-101.6	3								
C: Age 13 to 18								**C: Age 13 to 18**							
rostralmiddlefrontal	neg	61.42	0.0001	7.24E-08	-23.2	53.2	-4	rostralmiddlefrontal	neg	61.50	0.0001	4.13E-08	19.6	60.6	-11.4
isthmuscingulate	pos	17.89	0.0001	2.22E-07	-6.3	-50.5	17.9	lateraloccipital	pos	10.85	0.0064	5.19E-07	18.4	-101.3	-1.8
lateraloccipital	pos	10.92	0.0071	1.94E-05	-15.2	-102.8	-4.1	insula	pos	17.66	0.0001	1.10E-05	35.3	-14	12.3
Insula	pos	15.24	0.0002	2.01E-04	-30.3	-27.8	9.4	isthmuscingulate	pos	11.65	0.0038	2.43E-05	5.8	-48.9	19
superiorparietal	neg	9.00	0.0274	5.12E-04	-20.1	-84.5	22	postcentral	pos	8.58	0.0358	7.18E-05	15.1	-34.5	73

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD > ADHD, neg = ASD < ADHD;* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B8e: *T-contrast comparing the cortical thickness of ADHD and ASD combined to TDC*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
lateralorbitofrontal	pos	28.27	0.0001	6.53E-09	-10.1	49.6	-21.8	superiortemporal	neg	11.39	0.0042	1.69E-04	48.2	0.3	-25.2
rostralmiddlefrontal	pos	11.78	0.0045	1.36E-08	-37.5	36.9	8	superiorfrontal	pos	9.71	0.0155	6.44E-04	15.4	63	10.2
parahippocampal	neg	16.51	0.0001	3.39E-04	-21.7	-41.5	-8.9	lingual	pos	15.16	0.0003	3.61E-03	11.7	-90.9	-9.7
middletemporal	neg	8.71	0.0318	9.91E-04	-59	-21.3	-18.3								
lingual	pos	8.95	0.0277	1.06E-03	-5.4	-95.6	-12.2								
B: Age 8 to 12								**B: Age 8 to 12**							
lateralorbitofrontal	pos	27.30	0.0001	4.12E-08	-9.8	51.4	-21.3	parahippocampal	neg	10.93	0.0060	1.32E-05	25.5	-28.5	-21.2
rostralmiddlefrontal	pos	12.78	0.0021	6.49E-07	-37.4	38.2	7.3	parsopercularis	neg	8.89	0.0292	1.13E-04	52.7	13.3	7.2
precentral	pos	14.89	0.0003	5.21E-05	-43.1	-1.8	32.7								
lateraloccipital	pos	12.12	0.0031	1.24E-04	-14.9	-100.1	-11.8								
parahippocampal	neg	15.43	0.0001	3.04E-04	-22.7	-41.7	-8.6								
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								precentral	pos	12.16	0.0023	1.46E-04	32	-23.2	53.6

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD+ADHD > TDC, neg = ASD+ADHD < TDC;* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B9a: *F-contrast comparing the cortical surface area between all four groups*

Left Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages							**A: All ages**						
inferiortemporal	32.54	0.0001	5.77E-10	-53.6	-19.3	-25.3	middletemporal	27.98	0.0001	1.31E-07	55.1	-10.5	-24.5
cuneus	24.29	0.0003	4.12E-06	-20.7	-65.7	9.8	pericalcarine	50.74	0.0001	9.68E-07	14	-72	13.7
lateralorbitofrontal	44.96	0.0001	1.72E-05	-40.4	24.7	-13.6	superiorfrontal	24.67	0.0002	1.13E-04	8.4	61.3	-0.3
superiorparietal	17.23	0.0061	1.82E-04	-32.8	-41.1	53							
superiorparietal	20.69	0.0010	2.92E-03	-24.6	-72.5	23.7							
B: Age 8 to 12							**B: Age 8 to 12**						
inferiortemporal	28.50	0.0001	5.93E-08	-53.3	-18.8	-25.7	middletemporal	20.42	0.0012	2.40E-05	54.9	-14.8	-23.4
							medialorbitofrontal	22.50	0.0004	4.20E-05	7.7	25.1	-12.3
							pericalcarine	14.55	0.0217	6.43E-05	15.9	-73.7	12.4
C: Age 13 to 18							**C: Age 13 to 18**						
inferiortemporal	15.17	0.0145	3.85E-04	-38.9	-1.8	-41.1	lateraloccipital	48.98	0.0001	1.03E-04	16.1	-97.2	-9.2
lateraloccipital	20.66	0.0010	2.44E-03	-19.1	-99.2	-16	middletemporal	16.62	0.0093	6.61E-04	56.2	-8.4	-25
							inferiorparietal	18.30	0.0038	9.89E-04	36.4	-82.8	18.8

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B9b: *T-contrast comparing the cortical surface area of ADHD to TDC_{ADHD}*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
inferiortemporal	neg	23.68	0.0003	3.48E-05	-46.8	-16.1	-34.4	middletemporal	neg	27.14	0.0001	2.40E-05	53.3	-14.3	-24.5
rostralmiddlefrontal	neg	15.68	0.0118	2.51E-03	-36.2	39.9	15.4	pericalcarine	neg	15.54	0.0137	1.50E-04	7.1	-80.3	3.3
B: Age 8 to 12								**B: Age 8 to 12**							
inferiortemporal	neg	20.42	0.0011	2.82E-06	-42.8	-5.6	-41.7	inferiortemporal	neg	27.44	0.0001	9.79E-06	49.8	-12	-29.5
rostralmiddlefrontal	neg	20.10	0.0011	1.70E-04	-35.9	41.2	14	pericalcarine	neg	14.51	0.0223	1.23E-03	12.5	-84.3	1.3
C: Age 13 to 18								**C: Age 13 to 18**							
middletemporal	pos	13.56	0.0292	8.20E-04	-49.8	-63.6	-0.5	no cluster found							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ADHD > TDC_{ADHD}, neg = ADHD < TDC_{ADHD};* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B9c: *T-contrast comparing the cortical surface area of ASD to TDC_{ASD}*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
lingual	neg	19.92	0.0013	3.03E-04	-8.1	-97.9	-12.2	pericalcarine	neg	21.64	0.0007	3.43E-04	11.9	-73.7	14.5
B: Age 8 to 12								**B: Age 8 to 12**							
no cluster found								no cluster found							
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								pericalcarine	neg	18.80	0.0029	1.85E-03	12.8	-89.5	4.1

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. neg = ASD < TDC_{ASD};* **Size** = *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B9d: *T-contrast comparing the cortical surface area of ASD to ADHD*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
superiortemporal	pos	16.61	0.0079	4.79E-06	-52.6	-24.3	4.4	pericalcarine	neg	18.01	0.0045	1.71E-05	16.2	-70.8	12.2
parsorbitalis	pos	23.89	0.0003	2.65E-05	-40.6	27	-12.8	medialorbitofrontal	pos	19.92	0.0016	2.00E-04	11.3	54.6	1.2
middletemporal	neg	19.02	0.0026	9.04E-04	-54.4	-17.9	-22.9								
B: Age 8 to 12								**B: Age 8 to 12**							
superiortemporal	pos	18.90	0.0029	5.09E-07	-50.4	-22.9	4	medialorbitofrontal	pos	14.78	0.0194	3.85E-05	11	52.1	-1.4
								superiorfrontal	pos	13.49	0.0326	4.66E-04	20.2	47.8	34.4
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								no cluster found							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD > ADHD, neg = ASD < ADHD;* **Size** = *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B9e: *T-contrast comparing the cortical surface area of ADHD and ASD combined to TDC*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
pericalcarine	neg	19.24	0.0020	4.19E-04	-12.3	-83.1	3.9	pericalcarine	neg	25.98	0.0002	1.95E-05	10.9	-85.3	0.1
inferiorparietal	pos	15.54	0.0121	2.25E-03	-43.2	-64	11.1	fusiform	pos	12.50	0.0499	1.17E-04	31.6	-48.3	-12.7
								inferiortemporal	neg	15.11	0.0167	1.89E-03	37.3	2.2	-36.7
B: Age 8 to 12								**B: Age 8 to 12**							
inferiortemporal	neg	18.61	0.0031	3.61E-06	-41	-5.7	-42.8	pericalcarine	neg	13.48	0.0328	4.25E-03	12.6	-73.1	14.3
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								lingual	neg	13.22	0.0363	3.47E-03	6	-90.6	-10.3

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD+ADHD > TDC, neg = ASD+ADHD < TDC;* **Size** = *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B10a: *F-contrast comparing the cortical volume between all four groups*

Left Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages							**A: All ages**						
inferiortemporal	43.48	0.0001	4.99E-09	-40.7	-10.5	-34.8	lateraloccipital	30.18	0.0001	3.22E-08	19.9	-100.3	-3.4
lateraloccipital	36.04	0.0001	6.25E-08	-16	-102.6	-3.3	fusiform	32.29	0.0001	4.06E-07	36.7	-5.4	-41.6
lateralorbitofrontal	9.50	0.0333	4.35E-07	-8.7	55.8	-19.2	medialorbitofrontal	10.58	0.0177	7.06E-06	7.1	21.9	-10.5
inferiorparietal	11.94	0.0063	4.17E-05	-32	-76	22.6	paracentral	10.73	0.0155	2.84E-05	6.8	-21.3	56.6
precentral	9.28	0.0389	8.67E-05	-4.5	-37.7	67.7	lateralorbitofrontal	20.55	0.0001	7.94E-05	12.5	40.8	-19.9
							lateraloccipital	18.47	0.0001	2.25E-04	32.8	-82.4	13.3
B: Age 8 to 12							**B: Age 8 to 12**						
inferiortemporal	39.37	0.0001	1.08E-07	-41.2	-10.8	-35.2	medialorbitofrontal	15.68	0.0004	3.47E-07	7.2	22.5	-11.8
pericalcarine	11.69	0.0072	2.63E-04	-11.1	-86.9	3.6	inferiortemporal	19.87	0.0001	3.71E-06	55.2	-21.9	-22.6
C: Age 13 to 18							**C: Age 13 to 18**						
lateraloccipital	13.26	0.0033	7.40E-06	-19.9	-100.4	-7.7	lateraloccipital	33.43	0.0001	2.47E-07	20	-101	-0.4
precentral	9.37	0.0369	6.53E-05	-13.6	-20.2	69.1	parahippocampal	17.86	0.0001	3.67E-06	21.9	-32.3	-17.1
lingual	11.65	0.0074	8.45E-05	-12.3	-80.3	-8.1	lateralorbitofrontal	21.99	0.0001	7.73E-05	14.7	54.5	-15.7
lateralorbitofrontal	10.35	0.0201	1.91E-04	-8.9	58.8	-16.7	superiorparietal	16.83	0.0001	5.73E-04	24.4	-85.2	28.6
superiorparietal	11.71	0.0072	1.22E-03	-20.2	-69.8	38.5							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* $\mathbf{MNI_{X,Y,Z}}$ = *MNI-coordinate of MPC.*

B10b: *T-contrast comparing the cortical volume of ADHD to* TDC_{ADHD}

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
superiorfrontal	pos	12.90	0.0036	1.98E-06	-10.1	45.7	43.3	medialorbitofrontal	neg	13.07	0.0030	2.64E-07	6.8	21.1	-10.3
rostralanteriorcingulate	neg	11.05	0.0121	1.31E-05	-7.3	35	-1.3	fusiform	neg	26.69	0.0001	1.46E-05	36.7	-5.4	-41.6
inferiortemporal	neg	19.34	0.0001	1.14E-04	-43	-12.9	-32.6								
B: Age 8 to 12								**B: Age 8 to 12**							
inferiortemporal	neg	19.55	0.0001	4.08E-05	-39.7	-8.9	-41.5	medialorbitofrontal	neg	13.76	0.0019	1.84E-08	7.2	22.5	-11.8
fusiform	neg	9.32	0.0378	4.58E-05	-30.4	-36.2	-23.1	inferiortemporal	neg	26.15	0.0001	7.05E-07	55.3	-22.5	-22.3
C: Age 13 to 18								**C: Age 13 to 18**							
inferiortemporal	pos	12.74	0.0038	5.74E-04	-48.6	-60.6	-8.5	no cluster found							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ADHD >* TDC_{ADHD}*, neg = ADHD <* TDC_{ADHD}*;* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* $\mathbf{MNI_{X,Y,Z}}$ = *MNI-coordinate of MPC.*

B10c: *T-contrast comparing the cortical volume of ASD to TDC_{ASD}*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
pericalcarine	neg	12.72	0.0038	3.17E-04	-11.5	-85.9	3.4	pericalcarine	neg	15.31	0.0006	6.05E-05	14.2	-81.5	11
B: Age 8 to 12								**B: Age 8 to 12**							
pericalcarine	neg	8.92	0.0483	2.09E-04	-11.3	-85.8	2.4	no cluster found							
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								no cluster found							

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. neg = ASD < TDC_{ASD};* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B10d: *T-contrast comparing the cortical volume of ASD to ADHD*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z
A: All ages								**A: All ages**							
lateraloccipital	pos	10.23	0.0211	6.24E-06	-12.5	-103.5	-2.5	lateraloccipital	pos	10.60	0.0176	2.50E-07	19.3	-101.2	0.1
superiortemporal	pos	16.23	0.0004	1.09E-05	-55.1	-28.6	7.4	lateralorbitofrontal	neg	11.25	0.0107	4.52E-05	12.2	40.7	-19
middletemporal	neg	17.57	0.0001	1.92E-04	-55.3	-22.7	-21.4	inferiorparietal	neg	14.88	0.0009	1.76E-04	39.5	-79.3	14.9
inferiorparietal	neg	11.86	0.0065	1.93E-04	-32.2	-76.8	25	rostralmiddlefrontal	neg	9.78	0.0310	2.32E-03	37.1	34.2	17.9
B: Age 8 to 12								**B: Age 8 to 12**							
superiortemporal	pos	17.85	0.0001	7.57E-06	-55.1	-28.6	7.4	no cluster found							
C: Age 13 to 18								**C: Age 13 to 18**							
lateraloccipital	pos	14.84	0.0008	4.06E-05	-20	-100.6	-6.9	lateraloccipital	pos	21.21	0.0001	7.62E-08	19.9	-101.1	0.3
superiorparietal	neg	15.02	0.0008	1.16E-04	-20.1	-70.2	39.1	inferiorparietal	neg	19.13	0.0001	1.08E-03	33.7	-81.3	14.2

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD > ADHD, neg = ASD < ADHD;* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B10e: *T-contrast comparing the cortical volume of ADHD and ASD combined to TDC*

Left Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI_Z	**Right** Region	Eff.	Size [cm^2]	CWP	MPC	MNI_X	MNI_Y	MNI z
A: All ages								**A: All ages**							
rostralmiddlefrontal	pos	13.00	0.0035	1.18E-04	-21	37.3	34.7	pericalcarine	neg	12.85	0.0038	1.42E-06	15	-84.1	4.9
precuneus	pos	10.79	0.0145	1.21E-04	-8.2	-45.8	43.2	superiorfrontal	neg	11.99	0.0072	1.67E-04	14.4	36.4	16.4
B: Age 8 to 12								**B: Age 8 to 12**							
inferiortemporal	neg	20.74	0.0001	4.97E-05	-41.6	-11.1	-35.6	rostralanteriorcingulate	neg	16.13	0.0003	2.77E-05	12.3	37.8	11.2
paracentral	pos	9.93	0.0251	4.53E-04	-11.3	-40.8	72.3	pericalcarine	neg	9.56	0.0367	4.33E-05	15.3	-83.8	6
								precuneus	pos	10.92	0.0135	2.48E-04	8.5	-41.8	38.5
C: Age 13 to 18								**C: Age 13 to 18**							
no cluster found								lingual	pos	12.89	0.0037	1.35E-04	21.4	-58.8	-6.6

Region = *location of the cluster peak (peak adjacent regions are not listed);* **Eff.** = *direction of the effect. pos = ASD+ADHD > TDC, neg = ASD+ADHD < TDC;* **Size =** *size of the cluster on the surface model, shown in* cm^2*;* **CWP** = *cluster-wise p-value;* **MPC** = *maximum p-value in cluster;* **$MNI_{X,Y,Z}$** = *MNI-coordinate of MPC.*

B11a: *Summary of findings in this study concerning cortical and subcortical volume differences between ASD, ADHD and TDC.*

	ASD vs. TDC$_{ASD}$			***ADHD vs. TDC$_{AD}$***			***ASD vs. ADHD***			***ASD+ADHD vs. TDC***		
Volume	*All*	*young*	*old*	*All*	*young*	*Old*	*All*	*young*	*Old*	*All*	*young*	*Old*
Total gray matter volume				**dec.**	**dec.**		**inc.**	**inc.**		-	-	-
Total white matter volume										-	-	-
Frontal lobe										-	-	-
lateralorbitofrontal							***dec.$_r$***					
medialorbitofrontal				***dec.$_r$***	***dec.$_r$***					***dec.$_r$***	***dec.$_r$***	
parsopercularis							***dec.$_r$***					
rostralmiddlefrontal							***dec.$_r$***			***inc.$_l$***		
superiorfrontal				***inc.$_l$***						***inc.$_l$***		
paracentral										***inc.$_l$***	***inc.$_b$***	
Parietal lobe									**dec.**	-	-	-
postcentral											***inc.$_l$***	
supramarginal							***inc.$_l$***	***inc.$_l$***				
inferiorparietal							***dec.$_b$***		***dec.$_b$***			
superiorparietal							***dec.$_b$***		***dec.$_b$***			
precuneus										***inc.$_l$***	***inc.$_b$***	
Occipital lobe										-	-	-
pericalcarine	***dec.$_b$***	***dec.$_l$***									***dec.$_r$***	
Lingual	***dec.$_b$***	***dec.$_l$***						***inc.$_r$***		***dec.$_r$***		***inc.$_l$***
lateraloccipital							***inc.$_l$*** ***dec.$_r$***	***inc.$_b$***				
occipitalpole							***inc.$_r$***	***inc.$_b$***				
Temporal lobe				**dec.**	**dec.**					-	-	-
superiortemporal				***dec.$_b$***	***dec.$_r$***		***inc.$_l$***	***inc.$_l$***				
middletemporal				***dec.$_r$***	***dec.$_r$***		***dec.$_l$***					
inferiortemporal				***dec.$_b$***	***dec.$_b$***	***inc.$_l$***	***dec.$_l$***			***dec.$_l$***		
Fusiform				***dec.$_b$***	***dec.$_b$***					***dec.$_l$***		***inc.$_l$***
entorhinal				***dec.$_l$***	***dec.$_l$***					***dec.$_l$***		
temporalpole					***dec.$_l$***							
Cingulate cortex		**dec.**		**dec.**	**dec.**		**inc.**		**inc.**	-	-	-
rostralanteriorcingulate				***dec.$_b$***	***dec.$_r$***					***dec.$_r$***	***dec.$_r$***	
caudalanteriorcingulate				***dec.$_l$***								
posteriorcingualte											***dec.$_r$***	
Insular cortex				**dec.**	**dec.**					-	-	-
Corpus callosum				**inc.**	**inc.**					-	-	-
CC Central				**inc.**	**inc.**							
CC Mid-Anterior				**inc.**	**inc.**							
Subcortical regions										-	-	-
Accumbens Area		**inc.**					**inc.**	**inc.**	**inc.**	-	-	-
Amygdala							**inc.**		**inc.**	-	-	-
Basal Ganglai										-	-	-
Caudate							**inc.**	**inc.**		-	-	-
Pallidum						**dec.**				-	-	-
Putamen								**inc.**		-	-	-
Hippocampus									**inc.**	-	-	-
Thalamus Proper	**dec.**						**dec.**			-	-	-

Descriptive summary of analysis results conducted in this study, looking at cortical and subcortical volume differences; **Rows with gray background** *stem from ANCOVA analysis done on specific ROIs and hemispheres were not analyzed separately;* **Rows with white background** *show results from the vertex-wise analysis. Areas mentioned were at least partially responsible for the cluster found and were assed manually. Lateralization is indicated by* **b** *= bilateral,* **l** *= left and* **r** *= right;* **"-"** *= contrast was not calculated;* **All** *= analysis done with children between 8 and 18 years;* **young** *= analysis done with children between 8 to 12 years;* **old** *= analysis done with children between 13 and 18 years; The terms* **inc.** *= "increased" and* **dec.** *= "decreased" stands for the direction of the brain difference between the comparison groups.*

B11b: *Summary of findings in this study concerning cortical thickness differences between ASD, ADHD and TDC.*

	ASD vs. TDC_{ASD}			***ADHD vs. TDC_{AD}***			***ASD vs. ADHD***			***ASD+ADHD vs. TDC***		
Thickness	*All*	*young*	*old*	*All*	*young*	*old*	*All*	*Young*	*Old*	*All*	*young*	*old*
Mean cortical thickness							**inc.**		**inc.**	-	-	-
Frontal lobe				**inc.**	**inc.**		**dec.**	**dec.**	**dec.**	-	-	-
frontalpole					**inc.**$_b$		**dec.**$_b$	**dec.**$_l$	**dec.**$_b$			
lateralorbitofrontal				**inc.**$_b$	**inc.**$_l$		**dec.**$_b$	**dec.**$_r$	**dec.**$_b$	**inc.**$_l$	**inc.**$_l$	
medialorbitofrontal				**inc.**$_l$	**inc.**$_b$		**dec.**$_b$	**dec.**$_b$	**dec.**$_b$	**inc.**$_l$	**inc.**$_l$	
parsorbitalis							**dec.**$_b$		**dec.**$_l$			
parstriangularis					**inc.**$_l$					**inc.**$_l$	**inc.**$_l$	
parsopercularis				**dec.**$_l$	**dec.**$_l$		**dec.**$_r$		**dec.**$_l$		**dec.**$_b$	
rostralmiddlefrontal				**inc.**$_b$	**inc.**$_b$		**dec.**$_b$	**dec.**$_b$	**dec.**$_b$	**inc.**$_b$	**inc.**$_l$	
caudalmiddlefrontal					**inc.**$_l$		**dec.**$_b$		**dec.**$_l$		**inc.**$_l$	
superiorfrontal				**inc.**$_b$	**inc.**$_b$		**dec.**$_b$	**dec.**$_r$	**dec.**$_b$	**inc.**$_r$	**inc.**$_l$	
paracentral							**inc.**$_l$					
precentral				**dec.**$_l$	**dec.**$_l$		**inc.**$_b$	**inc.**$_l$			**inc.**$_l$ **dec.**$_r$	**inc.**$_r$
Parietal lobe										-	-	-
postcentral							**inc.**$_b$	**inc.**$_l$	**inc.**$_b$			**inc.**$_r$
supramarginal									**inc.**$_l$			
superiorparietal			**inc.**$_l$						**dec.**$_l$			
precuneus							**inc.**$_b$		**inc.**$_b$			
Occipital lobe				**inc.**			**inc.**		**inc.**	-	-	-
Cuneus							**inc.**$_l$					
pericalcarine				**inc.**$_b$	**inc.**$_r$		**inc.**$_l$					
Lingual				**inc.**$_b$	**inc.**$_b$		**inc.**$_l$			**inc.**$_b$		
lateraloccipital			**dec.**$_r$		**inc.**$_l$		**inc.**$_b$	**inc.**$_l$	**inc.**$_b$	**inc.**$_b$	**inc.**$_l$	
occipitalpole											**inc.**$_l$	
Temporal lobe	**dec.**			**dec.**	**dec.**					-	-	-
superiortemporal				**dec.**$_l$	**dec.**$_b$					**dec.**$_r$		
middletemporal	**dec.**$_l$			**dec.**$_r$	**dec.**$_r$							
inferiortemporal	**dec.**$_l$			**dec.**$_r$	**dec.**$_b$							
Fusiform				**dec.**$_b$	**dec.**$_b$					**dec.**$_l$	**dec.**$_b$	
parahippocampal				**dec.**$_l$	**dec.**$_b$					**dec.**$_l$	**dec.**$_b$	
entorhinal				**dec.**$_l$	**dec.**$_l$							
temporalpole					**dec.**$_b$							
Cingulate cortex				**dec.**	**dec.**		**inc.**		**inc.**	-	-	-
caudalanteriorcingulate							**inc.**$_l$					
posteriorcingualte							**inc.**$_b$		**inc.**$_l$			
isthmuscingualte							**inc.**$_b$		**inc.**$_b$			
Insular cortex				**dec.**	**dec.**	**dec.**	**inc.**		**inc.**	-	-	-
Insula				**dec.**$_l$	**dec.**$_r$		**inc.**$_b$		**inc.**$_b$			
Corpus callosum							**inc.**$_l$			-	-	-

Descriptive summary of analysis results conducted in this study, looking at cortical thickness differences; **Rows with gray background** *stem from ANCOVA analysis done on specific ROIs and hemispheres were not analyzed separately;* **Rows with white background** *show results from the vertex-wise analysis. Areas mentioned were at least partially responsible for the cluster found and were assed manually. Lateralization is indicated by* **b** *= bilateral,* **l** *= left and* **r** *= right;* **"-"** *= contrast was not calculated;* **All** *= analysis done with children between 8 and 18 years;* **young** *= analysis done with children between 8 to 12 years;* **old** *= analysis done with children between 13 and 18 years; The terms* **inc.** *= "increased" and* **dec.** *= "decreased" stands for the direction of the brain difference between the comparison groups.*

B11c: *Summary of findings in this study concerning cortical surface area differences between ASD, ADHD and TDC.*

	ASD vs. TDC$_{ASD}$			***ADHD vs. TDC$_{AD}$***			***ASD vs. ADHD***			***ASD+ADHD vs. TDC***		
Surface Area	*All*	*young*	*old*	*All*	*young*	*old*	*All*	*Young*	*Old*	*All*	*young*	*Old*
Total cortical surface area				**dec.**	**dec.**					-	-	-
Frontal lobe	**inc.**	**inc.**								-	-	-
frontalpole							***inc.$_r$***	***inc.$_r$***				
lateralorbitofrontal							***inc.$_l$***					
medialorbitofrontal							***inc.$_r$***	***inc.$_r$***				
parsorbitalis							***inc.$_l$***					
parstriangularis							***inc.$_l$***					
parsopercularis				***dec.$_l$***	***dec.$_l$***							
rostralmiddlefrontal				***dec.$_l$***	***dec.$_l$***							
superiorfrontal							***inc.$_r$***	***inc.$_r$***				
Parietal lobe				**inc.**	**inc.**		**dec.**		**dec.**	-	-	-
supramarginal							***inc.$_l$***	***inc.$_l$***				
inferiorparietal										***inc.$_l$***		
Occipital lobe	**dec.**							**dec.**		-	-	-
Cuneus	***dec.$_l$***		***dec.$_l$***	***dec.$_r$***			***dec.$_r$***			***dec.$_r$***		
pericalcarine	***dec.$_b$***		***dec.$_l$***	***dec.$_r$***	***dec.$_r$***		***dec.$_r$***			***dec.$_b$***	***dec.$_r$***	***dec.$_r$***
Lingual	***dec.$_b$***		***dec.$_l$***	***dec.$_r$***	***dec.$_r$***					***dec.$_b$***		***dec.$_r$***
lateraloccipital	***dec.$_b$***		***dec.$_l$***							***dec.$_l$***		
Temporal lobe					**dec.**					-	-	-
superiortemporal				***dec.$_r$***	***dec.$_r$***		***inc.$_l$***	***inc.$_l$***				
middletemporal				***dec.$_b$***	***dec.$_b$***	***inc.$_l$***	***dec.$_l$***			***inc.$_l$*** ***dec.$_r$***		
inferiortemporal				***dec.$_b$***	***dec.$_b$***	***inc.$_l$***	***dec.$_l$***			***inc.$_l$*** ***dec.$_r$***	***dec.$_l$***	
Fusiform				***dec.$_b$***	***dec.$_l$***					***inc.$_r$***	***dec.$_l$***	
parahippocampal										***inc.$_r$***		
entorhinal											***dec.$_l$***	
temporalpole				***dec.$_r$***						***dec.$_r$***		
Cingulate cortex					**dec.**					-	-	-
Insular cortex										-	-	-
Insula							***inc.$_l$***					

Descriptive summary of analysis results conducted in this study, looking at cortical surface area differences; **Rows with gray background** *stem from ANCOVA analysis done on specific ROIs and hemispheres were not analyzed separately;* **Rows with white background** *show results from the vertex-wise analysis. Areas mentioned were at least partially responsible for the cluster found and were assed manually. Lateralization is indicated by* **b** *= bilateral,* **l** *= left and* **r** *= right;* **"-"** *= contrast was not calculated;* **All** *= analysis done with children between 8 and 18 years;* **young** *= analysis done with children between 8 to 12 years;* **old** *= analysis done with children between 13 and 18 years; The terms* **inc.** *= "increased" and* **dec.** *= "decreased" stands for the direction of the brain difference between the comparison groups.*

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Selbständigkeitserklärung

Hiermit erkläre ich, dass die Masterarbeit von mir selbst und ohne unerlaubte Beihilfe verfasst worden ist und ich die Grundsätze wissenschaftlicher Redlichkeit einhalte (vgl. dazu: http://www.lehre.uzh.ch/index/LK-Plagiate-Merkblatt.pdf).

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Ort und Datum Unterschrift

CURRICULUM VITAE

MICHAEL NOTTER

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Nationality	Swiss

EDUCATION

UNIVERSITY OF ZURICH, Switzerland

02/2012 – 07/2014 **Master of Science in Cognitive Psychology and Cognitive Neuroscience**

Topics: neurobiology of psychological disorders, plasticity, sleep and memory, motivation and emotion, advanced analysis and methods of investigation technologies in neurophysiology, topographic analysis of EEG/ERP data

Minor Neuroinformatics

Topics: theory, programming and simulation of neural networks, models of computation, computational vision and foundational literature of Neuroscience

09/2007 – 02/2012 **Bachelor of Science in Psychology**

Topics: cognitive, clinical, biological, developmental, social and personality psychology, cognitive neuroscience, psychopathology, diagnostics, statistics, perception

Minor Neuroinformatics

Topics: Neuroscience (neurobiology of consciousness, introduction to Systems Neuroscience, from Networks to Systems), Informatics (Java, formal methods for Computer Science, introduction to Artificial Intelligence), Biology (Structure, Development, Plasticity and Repair of the Nervous System), Mathematics (analysis, linear algebra)

10/2006 - 02/2007 **ETH (SWISS FEDERAL INSTITUTE OF TECHNOLOGY) ZURICH**, Switzerland

1st semester of Bachelor of Science in Physics

Topics: analysis, linear algebra, physics, informatics and numerics

08/1999 - 07/2005 **ACADEMIC UPPER SECONDARY SCHOOL ZUG**, Switzerland

Swiss baccalaureate (university qualification) with majors in Mathematics and Physics and minor in Philosophy.

RESEARCH-RELATED EXPERIENCE

04/2014 - present **CHUV (CENTRE HOSPITALIER UNIVERSITAIRE VAUDOIS AND UNIVERSITY OF LAUSANNE)**, Lausanne, Switzerland
Position: research assistant
Work included: research in neuroscience and neuroimaging (operation of MRI scanning and EEG recording, implementation and maintenance of analysis software, design and implementation of experiments).

02/2013 - present **INAPIC (INTERNATIONAL NORMAL AGING AND PLASTICITY IMAGING CENTER)**, Zurich, Switzerland
Position: internship and research assistant
Work included: research in neuroscience (writing and maintaining of scripts for the analysis of behavioral data) and neuroimaging (operation of MRI scanning, implementation and maintenance of MRI analysis software, analysis of physiological data during fMRI) as well as extensive support of study staff with MRI analyses.

01/2011 - 05/2011 **MIT (MASSACHUSETTS INSTITUTE OF TECHNOLOGY)**, Cambridge, MA
Position: Internship at Gabrieli Lab, Department of Brain and Cognitive Sciences; prolonged to four months because of highly satisfactory work results.
Work included: research in neuroscience and neuroimaging (operation of MRI scanning, implementation and maintenance of analysis software, design and implementation of experiments), second author of the paper "A Cortico-Striatal Neural System Enhances Auditory Perception Through Temporal Context Processing " and assisting in other experiments by evaluation of results
Volunteer work: writing a comprehensive documentation for the newly developed neuroimaging software (miykael.github.com/nipype-beginner-s-guide/). Other programing tasks included adaptation of statistical normalization tools (ANTS) and teaching in Gabrieli Lab

WORK EXPERIENCE

03/2007 - 04/2014 **MIGROS BANK AG**, Zurich, Switzerland
Part time employment (40%) in Payment Transactions in the field of Preliminary Clarifications; Responsible for analyzing, creating and verifying team intern processes, assisting supervisor in various team matters and responsible for complex cases of the daily business by analyzing a customer's history or direct contact by phone or letter.

SWISS MILITARY, Switzerland
06/2012 - present Special officer in the psychological and pedagogical service of the army
03/2006 - 06/2012 Basic training with specialization as an anti-tank gunner

08/2005 - 02/2006 **SWISSCARD AECS**, Horgen, Switzerland
Temporary employment in Customer Service - Back-Office Credit Cards; Responsible for mutation and cancelation of VISA-Card, MasterCard and American Express clients and member of special unit "miles & more"

RESEARCH PUBLICATIONS & POSTERS

Geiser, E., Notter, M., & Gabrieli, J.D.E. (2012). A corticostriatal neural system enhances auditory perception through temporal context processing. *The Journal of Neuroscience, 32(18)*, 6177-6182.

Gorgolewski, K., Halchenko, Y., Hanke, M., Notter, M., Varoquaux, G., Waskom, M.L., Ziegler, E., Ghosh, S. (2012) Studying resting state connectivity using Nipype. *Poster at third biennial resting state conference, Magdeburg, Germany*, Sep 5-7.

Gorgolewski, K., Halchenko, Y., Hanke, M., Notter, M., Varoquaux, G., Waskom, M., Ziegler, E., Ghosh, S. (2012) Nipype 2012: more packages, reusable workflows and reproducible science. *Poster at 18th Annual Meeting of the Organization for Human Brain Mapping,* June 10-14.

LANGUAGES

German:	oral: fluent	written: fluent	mother tongue
English:	oral: fluent	written: fluent	
French:	oral: intermediate	written: intermediate	

COMPUTER SKILLS

Software: Windows, Linux, Mac OS, Microsoft Office, LaTeX, Sphinx (Python)
Languages: Bash, Java, Mathematica, MATLAB, Python, R
Neuroimagingsoftw.: Nipype, FreeSurfer, FSL, SPM, ANTs

EXTRACURRICULAR ACTIVITIES AND INTERESTS

Private tutor: Mathematics, physics and statistics for primary, high school and undergraduate students since 2003
Volunteer work: Writing summaries for 12 different bachelor courses (e.g. statistics, social psychology informatics, perception) and providing them as learning material to other students

REFERENCES

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Supervisor at MIT:	Eveline Geiser, Ph.D.	e-mail: egeiser@mit.edu
Supervisor at CHUV:	Micah M. Murray, Ph.D.	e-mail: micah.murray@chuv.ch