Psychiatry Research: Neuroimaging 289 (2019) 37–44

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

journal homepage: www.elsevier.com/locate/psychresns

Cortical structure abnormalities in females with conduct disorder prior to age 15

Meenal Budhiraja[a,*], Joana B. Pereira[b], Philip Lindner[a], Eric Westman[b], Jussi Jokinen[a,c], Ivanka Savic[d], Jari Tiihonen[a], Sheilagh Hodgins[a,e]

[a] Department of Clinical Neuroscience, Karolinska Institute, Psychiatry Building R5:00, Karolinska, University Hospital, Stockholm 171 76, Sweden
[b] Department of Neurobiology, Care Sciences and Society, Karolinska Institute, Stockholm, Sweden
[c] Department of Clinical Sciences/Psychiatry, Umeå University, Umeå, Sweden
[d] Department of Women's and Children's Health, Karolinska Institute, Stockholm, Sweden
[e] Département de Psychiatrie, Université de Montréal, Montreal, QC, Canada

ARTICLE INFO

Keywords:
Conduct disorder
Structural MRI
Freesurfer
Cortical thickness
Surface area
Fusiform gyrus

ABSTRACT

Among females, conduct disorder (CD) before age 15 is associated with multiple adverse outcomes in adulthood. The few existing structural neuroimaging studies of females with CD report abnormalities of gray matter volumes. The present study compared cortical thickness and surface area of young women with childhood/adolescent CD and healthy women to determine whether cortical abnormalities were present in adulthood and whether they were related to prior CD. Structural brain images from 31 women with CD and 25 healthy women were analyzed using FreeSurfer. Group differences between cortical thickness and surface area were assessed using cluster-wise corrections with Monte Carlo simulations. Women with prior CD, relative to healthy women, showed: (1) reduced cortical thickness in left fusiform gyrus extending up to entorhinal cortex and lingual gyrus; (2) reduced surface area in right superior parietal cortex; (3) increased surface area in left superior temporal gyrus, and right precentral gyrus. These differences remained significant after adjusting for past comorbid disorders, current symptoms of anxiety and depression, current substance use as well as maltreatment. The study suggests that among females, CD prior to age 15 is associated with cortical structure abnormalities in brain regions involved in emotion processing and social interaction.

1. Introduction

Conduct disorder (CD) is estimated to affect between 0.8% and 9.2% of girls prior to age 15 (Loeber et al., 2000). CD is associated with multiple negative outcomes including antisocial behaviour (ASB), limited academic achievement, low psychosocial functioning, physical and mental health problems (Odgers et al., 2008) and offspring with conduct problems (Jaffee et al., 2006). CD is a moderately heritable disorder of neuro-development (Rhee and Waldman, 2002) in which cognitive and affective-emotional processing deficits may persist into adulthood (Frick and Viding, 2009). Typically, females with CD experience maltreatment, many present anxiety and depression, and by mid-adolescence almost all misuse substances (Hodgins et al., 2018).

Previous structural magnetic resonance imaging (MRI) studies using voxel based morphometery (VBM) have reported abnormalities in grey matter volume (GMV) in several brain regions among females with CD, including anterior insula and right striatum (Fairchild et al., 2013), prefrontal cortex, supramarginal gyrus, angular gyrus (Dalwani et al., 2015). These studies, however, failed to take account of maltreatment and the typical array of disorders comorbid with CD. In our previous study, young women with prior CD (CDW) as compared to healthy women (HW) presented increased GMV in the left superior temporal gyrus (STG) and reduced GMV in lingual gyrus, hippocampus and anterior cingulate cortex. However, all the differences were associated with maltreatment, or current or past comorbid disorders (Budhiraja et al., 2017).

Abnormalities of GMV identified in previous VBM studies of females with CD may reflect differences in cortical thickness (CT), and surface area (SA) or a combination of these measures (Hutton et al., 2009). Neurons within the cerebral cortex are organized into ontogenetic columns that run perpendicular to the surface of the brain. The radial unit hypothesis of cortical development postulates that the cortical SA is driven by the number of columns, whereas CT is influenced by the number of cells within a column (Rakic, 2009). CT and SA are

* Corresponding author.
E-mail address: meenal.budhiraja@ki.se (M. Budhiraja).

https://doi.org/10.1016/j.pscychresns.2018.12.004
Received 7 November 2017; Received in revised form 4 December 2018; Accepted 6 December 2018
Available online 06 December 2018
0925-4927/

influenced by regionally distinct genetic factors (Pannizon et al.,2009). GMV, which is a composite of both these measures, is likely to be affected by some combination of these factors. CT and SA are more specific and biologically significant measure of neuroanatomical changes than GMV (Panizzon et al., 2009). Therefore, focusing on these two more specific measures of brain structure may reveal important and additional brain abnormalities associated with CD as well as clarify previously discrepant findings. The specificity in knowledge of structural neural abnormalities associated with CD will facilitate research into etiological factors.

The few existing studies of CT and SA among individuals with CD included all male or mixed gender samples and reported abnormalities in frontal and temporal regions (Fahim et al., 2011; Fairchild et al., 2015; Hyatt et al., 2012; Jiang et al., 2015; Wallace et al., 2014). A positive association between CT in the STG and the number of CD symptoms was observed among male adolescents (Chumachenko et al., 2015). In a recent study, CD was associated with cortical thinning in ventromedial prefrontal cortex in both sexes. Females with CD showed increased CT in supramarginal gyrus and reduced SA in superior frontal gyrus, compared with controls (Smaragdi et al., 2017).

There is no prior study of CT and SA among females with CD, even though evidence suggests that the clinical phenotype (Brennan and Shaw, 2013) and genotype (Sjöberg et al., 2007) differ in females and males, as do neural structures associated with ASB (Gur et al., 2002), and trajectories of brain development (Raznahan et al., 2011). Neural abnormalities associated with CD may be obscured by examining mixed-sex samples (Smaragdi et al., 2017). The present study aimed to further understanding of the neural correlates of CD in females, using surface based morphometry to estimate CT and SA in exploratory whole brain analyses, by taking account of maltreatment and comorbid disorders, and examining associations with psychopathy affective facet scores. We hypothesized that young women who had presented CD prior to age 15 would display abnormalities of CT and SA in brain regions implicated in emotion regulation and social cognition, primarily the prefrontal cortex and limbic and paralimbic regions. We examined the same sample (one HW different) as previously described (Budhiraja et al., 2017).

2. Method

2.1. Subjects

The sample included 46 women, aged, on average 23 years, 31 CDW diagnosed prior to age 15 and 25 HW with no history of CD, or criminal behavior, no current or past axis I or II disorders, other than two cases of past alcohol abuse. CDW were first assessed (Hodgins et al., 2007) in mid-adolescence when those 18 years or younger completed the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman et al., 1997) and those 18 or older the Structured Clinical Interview for DSM-IV(SCID I, SCID II) (First et al., 1990, 2002). The CDW were re-assessed six, 12 and 60 months later (Hodgins et al., 2010, 2014). Eighteen months later scanning began. Before the scan, all participants completed the SCID I and II, Edinburgh Handedness Inventory (Oldfield, 1971), Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993), Drug Use Disorders Identification Test (DUDIT) (Berman et al., 2005), Beck Anxiety Inventory (BAI) (Beck et al., 1988) and Beck Depression Inventory (BDI) (Beck et al., 1961). Physical and sexual abuse were assessed at baseline and each follow-up among the CDW, and prior to the scan among the HW using the Conflict Tactics Scale (Straus et al., 1996), the Sexual and Physical Abuse Questionnaire (Kooiman et al., 2002), MacArthur Community Violence Instrument (Steadman et al., 1998), and the Sexual Experience Survey (Koss et al., 1987). The sum of scores on the vocabulary and block design subtests of the Wechsler Intelligence Scale estimated IQ. CDW who were aged 16 or younger at the first assessment completed the Wechsler Intelligence Scale for Children – Third Edition (Wechsler, 1991), and all other participants completed the Wechsler Adult Intelligence Scale – Revised (Wechsler, 1997). In mid-adolescence, 25 of the CDW were assessed using the Psychopathy Checklist: Youth Version (PCL:YV) (Forth et al., 2003), and 6 of the CDW and the HW were assessed prior to the scan using the Psychopathy Checklist Screening Version (PCL:SV) (Hart et al., 1995). (For more detailed description of the sample and measures see Supplementary Material).

2.2. MRI Data

MRI scans were acquired using an axial T1-weighted sequence on a 3-Tesla MRI scanner (MR750 GE Healthcare, Milwaukee, Wisconsin) at Karolinska University Hospital using following parameters: 176 slices; thickness = 1 mm; voxel size = 1 × 1 × 1 mm; FOV = 24.0, Flip angle = 12^0, matrix = 240 × 240, TR = 7.9 ms, TI = 450 ms, and TE = 3.1 ms. Total scanning time was 6 min, 8 s. Images were inspected for clinically relevant abnormalities by a radiologist.

FreeSurfer image analysis suite version 5.3.0 (http://surfer.nmr.mgh.harvard.edu/) was employed to perform semi-automated cortical reconstruction (Dale et al., 1999). Using automated algorithms, images were transformed to standard space, segmented, normalized, and smoothed using a standard 10 mm kernel. All surfaces were visually inspected and segmentation errors or topological defects were manually corrected by manual edits to the surfaces, and adding control points. After these 3D surfaces were constructed, CT was estimated as the shortest distance from white surface to the pial surface at each surface vertex (Fischl and Dale, 2000). Cortical SA was estimated by computing the change in area of each triangle when mapped into spherical atlas space through allocating one third of the area of each triangle to each of its vertices (Winkler et al., 2012). Additionally, total intracranial volume (TIV) was extracted for each participant.

2.3. Statistical Analyses

Independent t tests and Fischer's exact tests were conducted, using the Statistical Package for Social Sciences (SPSS, version 22), to compare groups on socio-demographic and clinical characteristics. To assess differences of CT between CDW and HW, a general linear model (GLM) was estimated at each vertex across the cortical surface in FreeSurfer. Age was entered as a nuisance variable as age has an independent and negative association with CT (Sowell et al., 2007) and in small sample sizes a variable that does not significantly differentiate groups may confound results (Barnes et al., 2010). IQ was also entered as a nuisance variable as it is associated with CT (Karama et al., 2009), and with antisocial behavior (Murray and Farrington, 2010). Initially, CT was included as the dependent variable, group as a factor, and age as a nuisance variable. Next, CT was included as the dependent variable, group as a factor, and age and IQ as nuisance variables. Similarly, to assess differences in SA between CDW and HW, a GLM was estimated and SA was included as the dependent variable, group as a factor, and age and TIV as nuisance variables. Next, age, IQ, and TIV were entered as nuisance variables. We first used a cluster-forming threshold of $p < 0.05$ (two-tailed) and then to correct for multiple comparisons, we used Monte Carlo simulations (10.000 iterations, cluster-wise p-threshold < 0.05).

To determine whether observed significant differences in CT and SA between the CDW and HW were specific to CD, a series of analyses were conducted, controlling for past comorbid disorders, current symptoms of anxiety and depression and alcohol and drug use, and maltreatment. Surface-based Regions of Interest (ROIs) were created for brain areas where statistically significantly group differences in CT or SA were detected. The mean CT or SA at each cluster ROI for each subject was extracted. For each significant group difference, GLM were computed in SPSS. To determine whether observed group differences would remain after comparisons were adjusted for past comorbid disorders and maltreatment, three models were computed. All models of CT were

Table 1
Socio-demographic and clinical characteristics of participants.

	Conduct disorder (n = 31)	Healthy (n = 25)	Statistics
Mean (SD) age (years)	24.1 (2.7)	23.2 (3.3)	$t(54) = -0.98$ $p = 0.11$
Mean (SD) score handedness	87.6 (33.6)	92.0 (10.3)	$t(54) = 1.19$, $p = 0.282$
Mean (SD) number of conduct disorder symptoms prior to age 15	5.8 (2.8)	0	
Mean (SD) IQ scores	16.5 (3.9)	19.0 (2.6)	$t(53) = 3.16$, $p = 0.006$
% Completed high school	45	80	FET $p = 0.008$
Past comorbid disorders			
% Alcohol dependence	39	0	FET $p < 0.001$
% Drug dependence	42	0	FET $p < 0.001$
% Anxiety disorder	80	0	FET $p < 0.001$
% Depression disorder	67	0	FET $p < 0.001$
% Attention deficit hyperactive disorder	9.7	0	FET $p = 0.167$
Maltreatment			
% Physical abuse by parents	38	0	FET $p < 0.001$
% Sexual abuse	61	0	FET $p < 0.001$
At the time of scan			
% Not employed in past 2 years	58.6	0	FET, $p = 0.001$
% Alcohol dependence	3.2	0	FET $p = 0.554$
% Drug dependence	6.5	0	FET $p = 0.302$
% Anxiety disorder	32	0	FET $p = 0.001$
% Depression disorder	13	0	FET $p = 0.08$
% Antisocial personality Disorder	9.7	0	FET $p = 0.162$
Mean (SD) AUDIT scores	6.4 (4.06)	4.8 (3.3)	$t(54) = 1.61$ $p = 0.107$
Mean (SD) DUDIT scores	4.5 (8.41)	0.24 (0.83)	$t(54) = 2.76$ $p = 0.016$
Mean (SD) BAI scores	11.41(10.1)	4.96 (4.5)	$t(54) = 3.17$ $p = 0.005$
Mean (SD) BDI scores	13.77(14.1)	3.26 (4.5)	$t(54) = 3.90$ $p = 0.001$
% with children	48	12	FET $p = 0.004$
Mean (SD) score aggressive behavior last 6 months	0.87 (1.60)	0.12 (.439)	$t(54) = 2.48$ $p = 0.018$

FET = Fisher Exact Test
AUDIT: Alcohol Use Disorder Identification Test
DUDIT: Drug Use Disorder Identification Test
BAI: Beck Anxiety Inventory
BDI: Beck Depression Inventory

adjusted for age and IQ, and models of SA for age, IQ and TIV. In addition, model 1 adjusted for past alcohol and drug dependence, model 2 adjusted for past anxiety and depression disorders and model 3 for history of physical and sexual abuse. Since few of the participants presented comorbid disorders in adulthood, two additional models were computed to determine whether observed group differences between CDW and HW would remain after adjusting for current alcohol and drug use, anxiety and depression symptoms. Model 1 adjusted for current alcohol and drug use and model 2 for current anxiety and depression symptoms. Results were considered significant at $p < 0.05$.

Pearson correlations were computed within CDW to determine whether CT or SA of brain areas that differentiated CDW from HW were linked to the number of CD symptoms or to facet 2 PCL: YV scores, partialling out effects of age, IQ and TIV.

2.4. Ethics approval

The current study, and all previous waves of data collection, was approved by Regional Ethical Review Board in Stockholm.

3. Results

3.1. Clinical characterstics

As presented in Table 1, the CDW and HW were similar in age and handedness and differed in IQ and education. As is typical, proportionately more of CDW had acquired past diagnoses of comorbid disorders, and had reported physical and sexual abuse. At the time of scan, the CDW reported greater use of drugs, more anxiety and depression symptoms, and more aggressive behaviour than HW. More than half of the CDW had not been employed in a job in the previous two years.

3.2. Cortical thickness and surface area differences

As shown in Fig. 1 and Table 2, after adjusting for age, the CDW, relative to HW, showed significantly decreased CT in a large cluster located in left fusiform gyrus extending up to the entorhinal cortex and lingual gyrus and reduced thickness in a large cluster located in the left dorsolateral prefrontal cortex (DLPFC). After adjusting for age and IQ, the cluster at left DLPFC was no longer significant while the group difference in left fusiform gyrus cluster remained significant. As presented in Table 3, the group difference in CT in left fusiform gyrus remained significant after controlling for past alcohol and drug dependence, past anxiety and depression, maltreatment, current alcohol and drug use, and current symptoms of anxiety and depression.

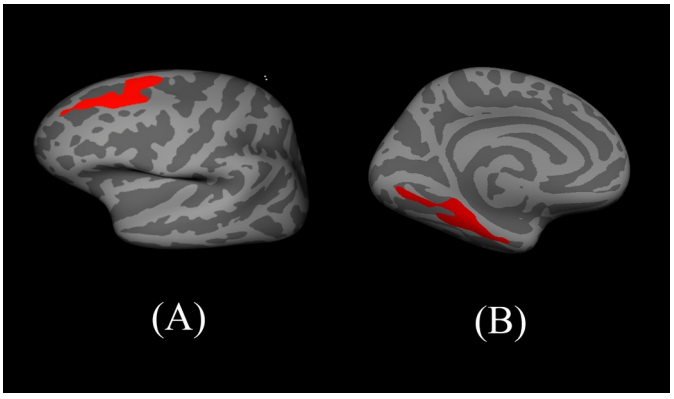

Fig. 1. (A) Reduced cortical thickness in left dorsolateral prefrontal cortex among women with conduct disorder prior to age 15 compared to the healthy women, adjusted for age. (B) Reduced cortical thickness in left fusiform gyrus of women with conduct disorder prior to age 15 compared to healthy women adjusted for age and IQ.

Table 2
Significant results of comparisons of cortical thickness of women with prior conduct disorder and healthy women.

	Brain region	Hemi-sphere	Number of verticies	Size (mm^2)	*X*	*Y*	*Z*	Maximum $-\log10$ (*P* value)	Cluster-wise-*P* value
Group comparisons after adjusting for age									
HW > CDW	Fusiform gyrus	Left	2482	1142.78	-31.3	-41.7	-12.8	5.889	0.02230
	Dorsolateral prefrontal cortex	Left	2137	1263.63	-22.4	19.4	47.5	2.389	0.01320
Group comparisons after adjusting for age and IQ									
HW > CDW	Fusiform gyrus	Left	2754	1461.43	-35.1	-23.3	-22.8	2.387	0.030

CDW: women with conduct disorder prior to age 15; HW: healthy women

As presented in Fig. 2 and Table 4, after adjusting for age, IQ, and TIV, the CDW, relative to HW, presented significantly decreased SA in a large cluster located at the right superior parietal cortex. CDW, relative to HW, displayed increased SA in left STG, right precentral cortex and at a large cluster in right rostral prefrontal cortex extending up to the lateral orbitofrontal cortex (OFC). As presented in Table 3, the group differences in SA in right superior parietal cortex, in left STG, and in the right precentral gyrus, remained significant after controlling for all covariates. However, the difference in the right prefrontal gyrus disappeared after controlling for past anxiety and depression disorders.

3.3. Correlation with CD symptoms and affective facet psychopathy scores

After controlling for age and IQ, none of the partial correlations between CT, SA and number of CD symptoms, and CT, SA, and psychopathy affective facet scores were significant.

4. Discussion

Young women who had presented CD before age 15, relative to HW, displayed reduced CT in left fusiform gyrus, decreased SA in right superior parietal cortex, and increased SA in left STG, and right precentral gyrus. Each of these group differences was robust to adjustment for age and IQ, physical and sexual abuse, past alcohol and drug dependence and anxiety and depression disorders, and current alcohol and drug use and anxiety and depression symptoms, suggesting that these abnormalities were specifically associated with prior CD. The CDW, relative to the HW, also showed reduced CT in the DLPFC when controlling for age and IQ, but not when controlling for age, and increased SA in the right rostral prefrontal cortex that was associated with past anxiety and depression disorders.

CT deficits in left fusiform gyrus were previously observed among adolescents with CD (Hyatt et al., 2012), and a meta-analysis reported reduced GMV in the left fusiform gyrus of youth with CD (Rogers and De Brito, 2016). The CDW displayed reduced SA in right superior parietal cortex. Reductions in CT (Hyatt et al., 2012) and decreased functional activity (White et al., 2012), is reported among youth with CD in this region. Increased SA in left STG was observed among the CDW, consistent with recent study of adult males with prior CD (Jiang et al., 2016). The increased SA in STG is also congruent with findings of increased GMV in left STG among adult males with ASB (Schiffer et al., 2013) and boys with conduct problems (De Brito et al., 2009). Increased SA in right precentral cortex was observed among the CDW relative to the HW, consistent with previous reports of structural (Jiang et al., 2016) and functional abnormalities in this region (Shannon et al., 2011) in individuals with ASB.

The left fusiform gyrus, superior parietal lobe, precentral gyrus and STG are crucial for social interaction as they play important roles in empathy (Hooker et al., 2010), moral reasoning (Raine and Yang, 2006), and recognizing emotions in faces, which is deficient in females with CD (Dadds et al., 2006; Fairchild et al., 2010). The identification of emotions in faces is a complex task that requires visual scanning, perceptual processing, effortful attention, working memory, and semantic processing (Marsh and Blair, 2008). This processing of emotions relies on a large, distributed network of neural structures of occipito-temporal visual cortex including the fusiform gyrus, superior parietal cortex, and STG (Labar et al., 2003). The fusiform gyrus is connected to the amygdala (Pujol et al., 2009), and plays a central role in social categorization by modulating face perception (Sabatinelli et al., 2011; Schwarz et al., 2013). The STG is important for efficient social cognition (Adolphs, 2003). The fusiform and STG are engaged to construct a spatial representation of facial features and movements (Haxby et al., 2000). The superior parietal cortex is critically important for the manipulation of information in working memory (Koenigs et al., 2009). The differences observed in the present study between the CDW and HW in the left fusiform gyrus, superior parietal lobe, precentral gyrus and STG could be related to deficits in emotion processing previously shown to characterize individuals with ASB (Marsh and Blair, 2008). A recent study showed, however, that among children with elevated callous-unemotional traits, only those without comorbid anxiety or who had not been maltreated presented deficient emotion recognition (Dadds et al., 2018). In the present study, neither the numbers of CD symptoms nor the PCL affective facet scores were correlated with CT and SA. Yet, the group differences remained after controlling for age, IQ, TIV, past mental disorders, current substance use, anxiety and depression. Future studies are needed to simultaneously assess emotion processing and structural abnormalities in children/adolescents with CD, taking account of comorbid disorders, maltreatment, and CU traits.

Females with CD displayed abnormalities in CT and SA, some of which are similar to cortical abnormalities observed in males or mixed/sex samples with CD (Hyatt et al., 2012; Sarkar et al., 2015). We also found novel evidence for SA abnormalities in superior parietal cortex among females with CD. These cortical abnormalities were observed at an average age of 24 years, when few of these CDW met the diagnostic criteria for antisocial personality disorder, yet, CDW displayed increased aggressive behavior, low levels of psychosocial functioning and educational attainment, and one-third presented anxiety disorders. These findings highlight the importance of intervening early to prevent childhood disorders, to promote healthy development, and to limit the inter-generational transfer of ASB.

The findings in the present study are partially consistent with our previous VBM study (Budhiraja et al., 2017). VBM analyses identified increased GMV in the left STG and here we found greater SA in left STG; VBM analyses identified reduced GMV in lingual gyrus and in the present study the CDW showed reduced CT in fusiform gyrus extending up to lingual gyrus. The current study demonstrates the value of applying surface-based morphometry methods to investigate neural correlate of CD, as alterations in CT and SA were not restricted to the cortical regions identified in our previous VBM study. Additionally, CDW, relative to the HW, showed reduced SA in the right superior parietal cortex, and increased SA in right precentral cortex. These cortical abnormalities were robust to all covariates, suggesting that they were directly associated with CD prior to age 15. By contrast, in our previous VBM study group differences were associated with maltreatment, past externalizing and internalizing disorders, and current

Table 3
Comparisons of cortical thickness and surface area between women with prior conduct disorder and healthy women corrected for maltreatment, past comorbid disorders, current alcohol and drug use, and current anxiety and depression symptoms.

Brain regions	Past alcohol and drug dependence			Past anxiety and depression			History of maltreatment			Current alcohol and drug use			Current anxiety and depression symptoms		
	df	*F*	*p*	*df*	*F*	*p*	*df*	*F*	*p*	*df*	*F*	*p*	*df*	*F*	*p*
CT fusiform gyrus	1,49	17.43	< 0.001	1,49	9.83	0.003	1,49	14.06	< .001	1,49	19.73	< 0.001	1,49	20.69	< 0.001
SA superior parietal cortex	1,49	10.72	0.002	1,49	4.50	0.039	1,49	4.23	0.045	1,49	11.75	0.001	1,49	7.57	0.008
SA rostral prefrontal cortex	1,49	7.36	0.009	1,49	2.14[a]	0.141	1,49	5.092	0.029	1,49	6.21	0.016	1,49	5.92	0.019
SA precentral gyrus	1,49	10.06	0.002	1,49	4.5	0.039	1,49	5.26	0.026	1,49	17.57	< 0.001	1,49	8.16	0.006
SA superior temporal gyrus	1,49	11.46	0.001	1,49	9.23	0.008	1,49	6.86	0.012	1,49	14.06	< 0.001	1,49	8.62	0.005

The dependent variables (extracted cortical thickness and surface area of significant brain region in whole brain analyses) are shown in the first column. All comparisons of surface area are corrected for age, IQ, and intracranial volume.
SA: surface area; CT: cortical thickness.
[a] not significant ($p > 0.05$)

substance use and anxiety and depression. The inconsistency in findings in our study using VBM and the present one reflect, at least in part, differences in the methodologies of the analysis techniques. FreeSurfer uses a surface-based morphometry approach compared with VBM's voxel-wise registration. Moreover, GMV is a composite of CT and SA, and consequently, within one region, a minor change of SA accompanied by a minor change in CT could result in significant change in the effect when expressed as GMV. Conversely, effects in opposite directions (increased SA / decreased CT) may cancel each other out and GMV may not show overlap with either measure.

The CDW, relative to the HW, displayed reduced CT, and increased SA. Similar results of thinner cortex and increased SA in several brain regions, including frontal gyrus, OFC, insula, precuneus, and left STG have been demonstrated in males with ASB (Jiang et al., 2016). There are complex regional patterns of associations between changes in SA and CT in the cortex during adolescence, with positive associations observed in prefrontal and temporal cortices, and negative associations observed mainly in more posterior cortices. These observations of increased SA in prefrontal and temporal cortices among females with CD may reflect delayed brain development (Oostermeijer et al., 2016).

The reduced CT in the DLPFC observed in the CDW was associated with IQ, consistent with a previous report (Karama et al., 2009). Individuals with CD typically present lower than average IQ (Murray and Farrington, 2010) and behaviors such as impulsivity, poor judgment, and difficulty making decisions, that are associated with DLPFC damage (Manes et al., 2002; Loveland et al., 2001). The CDW also displayed increased SA in right prefrontal cortex extending up to the OFC that was associated with past anxiety and depression disorders, consistent with a finding of increased CT in right prefrontal cortex among individuals with anxiety disorders (Brühl et al., 2014). Results of the present study suggest that abnormalities in CT and SA of prefrontal cortex that have been reported in individuals with CD in predominately male samples (Hyatt et al., 2012; Sarkar et al., 2015), may not be directly associated with CD design.

In order to identify the underlying mechanism of structure neural alterations associated with CD among females, it is essential to conduct prospective longitudinal studies that repeatedly assess disorders and brain structures and functioning. Among girls, genes increased the risk of conduct problems, other externalizing and internalizing problems (Rhee et al., 2015; Bloningen et al., 2005). Combinations of these problems emerge by age 3, and have been shown to be stable to age 15 (Fanti and Kimonis, 2017). Studies of gene-environment interplay show that CD genetic predispositions, interacting with environmental factors early in life lead to abnormal neural development that promote these problems (Salvatore and Dick, 2016). For example, the mono-amine A oxidase gene that has been associated with ASB and internalizing problems in females (Byrd and Manuck, 2014), acts primarily in the prenatal and early post-natal period to set levels of serotonergic activity (Harro and Oreland, 2016). Environmental factors that impact the girl subsequently, such as maltreatment, would have distinct effects on the brain depending on the level of serotonergic activity and some would lead to epigenetic changes thereby altering the expression of genes (Labonté et al., 2012) that may further promote abnormal brain structure and function. Additionally, the development of CT across the lifespan is largely determined by genes (Fjell et al., 2015). Thus, future genetically informed, longitudinal studies are needed to disentangle onset of CD and comorbid disorders and correlated neural structural development.

4.1. Limitations and strengths

Limitations included the small sample size cross-sectional design, and use of only two subtests to assess IQ. Strengths included a female-only sample that was typical of clinical samples, rich clinical characterization of participants obtained using validated measures that

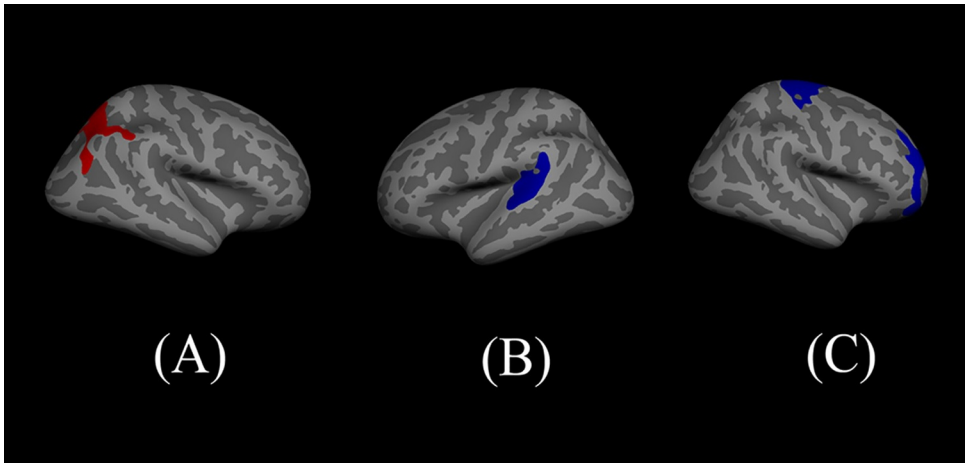

Fig. 2. (A) Reduced surface area in right superior parietal cortex of women with conduct disorder prior to age 15 compared to healthy women, adjusted for age, IQ and total intracranial volume. (B) Increased surface area in left superior temporal gyrus of women with conduct disorder prior to age 15 compared to healthy women, adjusted for age, IQ and total intracranial volume. (C) Increased surface area in right prefrontal cortex and precentral gyrus of women with conduct disorder prior to age 15 compared to healthy women, adjusted for age, IQ and total intracranial volume.

Table 4
Significant results of comparisons of surface area of women with prior conduct disorder and healthy women adjusted for age, IQ and total intracranial volume.

	Brain region	Hemisphere	Number of vertices	Size (mm^2)	*X*	*Y*	*Z*	Maximum $-\log10$ (*P* value)	Cluster-wise-*P* value
HW > CDW	Superior parietal corTEX	Right	4272	2126.75	27.8	-54.7	40.3	4.512	0.00820
HW < CDW	Rostral prefrontal cortex	Right	2783	1891.02	27.7	57.8	-9.5	2.389	0.01320
	Precentral gyrus	Right	3916	1695.40	13.9	19.8	71.9	1.4763	0.0300
	Superior temporal gyruS	Left	4497	1925.60	-41.2	-35.8	10	4.564	0.0149

CDW: women with conduct disorder prior to age 15; HW: healthy women

allowed adjustment of group comparisons for maltreatment and comorbid disorders. Stringent statistical thresholds were applied and group differences were found in large clusters, adding confidence to findings.

4.2. Conclusion

Among young adult women, CD prior to age 15 was associated with reduced CT in left fusiform gyrus, decreased SA in right superior parietal cortex, and increased SA in left STG and right precentral gyrus. These group differences were robust to adjustment for maltreatment, past externalizing and internalizing disorders, current substance use and anxiety and depression symptoms suggesting that they were directly associated with prior CD. The study suggests that among females, CD prior to age 15 is associated with cortical abnormalities in brain regions involved in emotion processing and social cognition.

Funding and disclosure

This research was funded by a grant from Mobilisering mot narkotika (Swedish National Drug Policy Coordinator) and from Stockholm Country Council. Authors Meenal Budhiraja, Philip Lindner and Jari Tiihonen are supported by a grant received from Swedish Foundation for Strategic Research. Author Ivanka Savic is supported by a grant from VINNOVA; Jussi Jokinen by grants received from Swedish Research Council and Regional Agreement on Medical Training and Clinical Research between Stockholm County Council and Karolinska Institute and Sheilagh Hodgins is supported by a grant from Stockholm County Council. Philip Lindner received honoraria from Pearson Assessments. Jari Tiihonen received consultancy fees from Lundbeck, Organon, Janssen -Cilag, Eli Lilly, AstraZeneca, F. Hoffman -La Roche, and Bristol -Myers Squibb; lecture fees from Janssen -Cilag, Bristol -Myers Squibb, Eli Lilly, Pfizer, Lundbeck, GlaxoSmithKline, AstraZeneca and Novartis; fees for expert opinions from Bristol -Myers Squibb and GlaxoSmithKline; and research funding from Stanley Foundation. Jari Tiihonen is also a member of advisory boards for AstraZeneca, Janssen -Cilag, and Otsuka.

Conflict of Interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Acknowledgments

We thank young women, who participated in the study.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.pscychresns.2018.12.004.

References

Adolphs, R., 2003. Cognitive neuroscience of human social behaviour. Nat. Rev. Neurosci. 4, 165–178. https://doi.org/10.1038/nrn1056.
Barnes, J., Ridgway, G.R., Bartlett, J., Henley, S., Lehmann, M., Hobbs, N., et al., 2010. Head size, age and gender adjustment in MRI studies: a necessary nuisance. Neuroimage 53, 1244–1255.
Beck, A.T., Epstein, N., Brown, G., Steer, R.A., 1988. An inventory for measuring clinical anxiety: psychometric properties. J. Consult. Clin. Psychol. 56, 893–897.
Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., Erbaugh, J., 1961. An inventory for measuring depression. Arch. Gen. Psychiatry 4, 561–571.
Berman, A.H., Bergman, H., Palmstierna, T., Schlyter, F., 2005. Evaluation of the Drug Use Disorders Identification Test (DUDIT) in criminal justice and detoxification settings and in a Swedish population sample. Eur. Addict. Res. 11, 22–31. https://doi.org/10.1159/000081413.
Bloningen, D.M., Hicks, B.M., Kruger, R., Patrick, C.J., Iacono, W.G., 2005. Psychopathic personality traits: heritability and genetic overlap with internalizing and externalizing psychopathology. Psychol. Med. 35, 637–648.
Brennan, L.M., Shaw, D.S., 2013. Revisiting data related to the age of onset and developmental course of female conduct problems. Clin. Child Fam. Psychol. Rev. 16, 35–58. https://doi.org/10.1007/s10567-012-0125-8.
Brühl, A.B., Delsignore, A., Komossa, K., Weidt, S., 2014. Neuroimaging in social anxiety

disorder—A meta-analytic review resulting in a new neurofunctional model. Neurosci. Biobehav. Rev. 47, 260–280. https://doi.org/10.1016/j.neubiorev.2014.08.003.
Budhiraja, M., Savic, I., Lindner, P., Jokinen, J., Tiihonen, J., Hodgins, S., 2017. Brain structure abnormalities in young women who presented conduct disorder in childhood/adolescence. Cognit. Affect. Behav. Neurosci. 17, 869–885. https://doi.org/10.3758/s13415-017-0519-7.
Byrd, A.L., Manuck, S.B., 2014. MAOA, childhood maltreatment, and antisocial behavior: meta-analysis of a gene-environment interaction. Biol. Psychiatry 75, 9–17.
Chumachenko, S.Y., Sakai, J.T., Dalwani, M.S., Mikulich-Gilbertson, S.K., Dunn, R., Tanabe, J., Young, S., McWilliams, S.K., Banich, M.T., Crowley, T.J., 2015. Brain cortical thickness in male adolescents with serious substance use and conduct problems. Am. J. Drug Alcohol Abuse 41, 414–424. https://doi.org/10.3109/00952990.2015.1058389.
Dadds, M.R., Perry, Y., Hawes, D.J., Merz, S., Riddell, A.C., Haines, D.J., Solak, E., Abeygunawardane, A.I., 2006. Attention to the eyes and fear-recognition deficits in child psychopathy. Br. J. Psychiatry 189, 280–281. https://doi.org/10.1192/bjp.bp.105.018150.
Dadds, M.R., Kimonis, E.R., Schollar-Root, O., Moul, C., Hawes, D.J., 2018. Are impairments in emotion recognition a core feature of callous–unemotional traits? Testing the primary versus secondary variants model in children. Dev. Psychopath 30, 67–77.
Dale, A.M., Fischl, B., Sereno, M.I., 1999. Cortical surface-based analysis: I. Segmentation and surface reconstruction 9, 179–194. https://doi.org/10.1006/nimg.1998.0395.
Dalwani, M.S., McMahon, M.A., Mikulich-Gilbertson, S.K., Young, S.E., Regner, M.F., Raymond, K.M., McWilliams, S.K., Banich, M.T., Tanabe, J.L., Crowley, T.J., Sakai, J.T., 2015. Female adolescents with severe substance and conduct problems have substantially less brain gray matter volume. PLOS One 10, e0126368. https://doi.org/10.1371/journal.pone.0126368.
De Brito, S.A., Mechelli, A., Wilke, M., Laurens, K.R., Jones, A.P., Barker, G.J., Hodgins, S., Viding, E., 2009. Size matters: increased grey matter in boys with conduct problems and callous-unemotional traits. Brain 132, 843–852. https://doi.org/10.1093/brain/awp011.
Fahim, C., He, Y., Yoon, U., Chen, J., Evans, A., Pérusse, D., 2011. Neuroanatomy of childhood disruptive behavior disorders. Aggress. Behav. 37, 326–337. https://doi.org/10.1002/ab.20396.
Fairchild, G., Hagan, C.C., Walsh, N.D., Passamonti, L., Calder, A.J., Goodyer, I.M., 2013. Brain structure abnormalities in adolescent girls with conduct disorder. J. Child Psychol. Psychiatry 54, 86–95. https://doi.org/10.1111/j.1469-7610.2012.02617.x.
Fairchild, G., Stobbe, Y., van Goozen, S.H.M., Calder, A.J., Goodyer, I.M., 2010. Facial expression recognition, fear conditioning, and startle modulation in female subjects with conduct disorder. Biol. Psychiatry 68, 272–279. https://doi.org/10.1016/j.biopsych.2010.02.019.
Fairchild, G., Toschi, N., Hagan, C.C., Goodyer, I.M., Calder, A.J., Passamonti, L., 2015. Cortical thickness, surface area, and folding alterations in male youths with conduct disorder and varying levels of callous-unemotional traits. Neuroimage Clin. 30, 253–260. https://doi.org/10.1016/j.nicl.2015.04.018.
Fanti, K.A., Kimonis, E., 2017. Heterogeneity in externalizing problems at Age 3: association with age 15 biological and environmental outcomes. Dev. Psychol. 53 (7), 1230–1241. https://doi.org/10.1037/dev0000317.
First, M.B., Gibbon, M., Spitzer, R.L., Williams, J.B.W., Benjamin, L.S, 1990. Structured Clinical Interview for DSM-IV Axis II Personality Disorders, (SCID-II). American Psychiatric Press, Inc, Washington, D.C.
First, M.B., Spitzer, R.L., Gibbon., M., Williams, J.B.W., 2002. Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition (SCID-I/P). Biometrics Research. New York State Psychiatric Institute, New York.
Fischl, B., Dale, A.M., 2000. Measuring the thickness of the human cerebral cortex from magnetic resonance images. PNAS 97, 11050–11055. https://doi.org/10.1073/pnas.200033797.
Fjell, A.M., Grydeland, H., Krogsrud, S.K., Amlien, I., Rohani, D.A., Ferschmann, L., et al., 2015. Development and aging of cortical thickness correspond to genetic organization patterns. PNAS 112 (50), 15462–15467. https://doi.org/10.1073/pnas.1508831112.
Forth, A.E., Kosson, D.S., Hare, R.D., 2003. Hare Psychopathy Checklist: Youth Version (PCL:YV): Technical Manual. Multi-Health Systems, Toronto.
Frick, P.J., Viding, E., 2009. Antisocial behavior from a developmental psychopathology perspective. Dev. Psychopathol. 21, 1111. https://doi.org/10.1017/S0954579409990071.
Gur, R.C., Gunning-Dixon, F., Bilker, W.B., Gur, R.E., 2002. Sex differences in temporolimbic and frontal brain volumes of healthy adults. Cereb. Cortex 12, 998–1003.
Hart, S.D., Cox, D., Hare, R.D., 1995. Manual for the Psychopathy Checklist: Screening Version (PCL: SV). Multi-Health Systems, Toronto.
Harro, J., Oreland, L., 2016. The role of MAO in personality and drug use. Prog. Neuropsychopharmacol. Biol. Psychiatry 69, 101–111. https://doi.org/10.1016/j.pnpbp.2016.02.013.
Haxby, J., Hoffman, E., Gobbini, M., 2000. The distributed human neural system for face perception. Trends Cogn. Sci. 4, 223–233. https://doi.org/10.1016/S1364-6613(00)01482-0.
Hodgins, S., Checknita, D., Lindner, P., Schiffer, B., De Brito, S., 2018. Antisocial personality disorder. In: Beech, A., Carter, A.J., Mann, R., Rotshtein, P. (Eds.), Handbook of Forensic Neuroscience. Wiley, Oxford, U.K..
Hodgins, S., Lövenhag, S., Rehn, M., Nilsson, K.W., 2014. A 5-year follow-up study of adolescents who sought treatment for substance misuse in Sweden. Eur. Child Adolesc. Psychiatry 23, 347–360. https://doi.org/10.1007/s00787-013-0456-0.
Hodgins, S., Oliver, B.R., Tengström, A., Larsson, A., 2010. Adolescents who consulted for substance misuse problems: outcomes 1 year later. Nord. J. Psychiatry 64, 189–195. https://doi.org/10.3109/08039480903389002.
Hodgins, S., Tengström, A., Bylin, S., Göranson, M., Hagen, L., Janson, M., Larsson, A., Lundgren-Andersson, C., Lundmark, C., Norell, E., Pedersen, H., 2007. Consulting for substance abuse: mental disorders among adolescents and their parents. Nord. J. Psychiatry 61, 379–386. https://doi.org/10.1080/08039480701643423.
Hooker, C.I., Verosky, S.C., Germine, L.T., Knight, R.T., D'Esposito, M., 2010. Neural activity during social signal perception correlates with self-reported empathy. Brain Res. 1308, 100–113. https://doi.org/10.1016/j.brainres.2009.10.006.
Hutton, C., Draganski, B., Ashburner, J., Weiskopf, N., 2009. A comparison between voxel-based cortical thickness and voxel-based morphometry in normal aging. Neuroimage 48, 371–380. https://doi.org/10.1016/j.neuroimage.2009.06.043.
Hyatt, C.J., Haney-Caron, E., Stevens, M.C., 2012. Cortical thickness and folding deficits in conduct-disordered adolescents. Biol. Psychiatry 72, 207–214. https://doi.org/10.1016/j.biopsych.2011.11.017.
Jaffee, S.R., Belsky, J., Harrington, H., Caspi, A., Moffitt, T.E., 2006. When parents have a history of conduct disorder: how is the caregiving environment affected. J. Abnorm. Psychol. 115, 309–319. https://doi.org/10.1037/0021-843X.115.2.309.
Jiang, W., Li, G., Liu, H., Shi, F., Wang, T., Shen, C., 2016. Reduced cortical thickness and increased surface area in antisocial personality disorder. Neuroscience 337, 143–152. https://doi.org/10.1016/j.neuroscience.2016.08.052.
Jiang, Y., Guo, X., Zhang, J., Gao, J., Wang, X., Situ, W., et al., 2015. Abnormalities of cortical structures in adolescent-onset conduct disorder. Psychol. Med. 45, 3467–3479. https://doi.org/10.1017/S0033291715001361.
Karama, S., Ad-Dab'bagh, Y., Haier, R.J., Deary, I.J., Lyttelton, O.C., Lepage, C., Evans, A.C., Brain Development Cooperative Group, B.D.C., 2009. Positive association between cognitive ability and cortical thickness in a representative US sample of healthy 6 to 18 year-olds. Intelligence 37, 145–155. https://doi.org/10.1016/j.intell.2008.09.006.
Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., Williamson, D., Ryan, N., 1997. Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): initial reliability and validity data. J. Am. Acad. Child Adolesc. Psychiatry 36, 980–988. https://doi.org/10.1097/00004583-199707000-00021.
Koenigs, M., Barbey, A.K., Postle, B.R., Grafman, J., 2009. Superior parietal cortex is critical for the manipulation of information in working memory. J. Neurosci. 29, 14980–14986. https://doi.org/10.1523/JNEUROSCI.3706-09.2009.
Kooiman, C.G., Ouwehand, A.W., ter Kuile, M.M., 2002. The Sexual and Physical Abuse Questionnaire (SPAQ). A screening instrument for adults to assess past and current experiences of abuse. Child Abuse Negl. 26, 939–953. https://doi.org/10.1016/S0145-2134(02)003630.
Koss, M.P., Gidycz, C.A., Wisniewski, N., 1987. The scope of rape: incidence and prevalence of sexual aggression and victimization in a national sample of higher education students. J. Consult. Clin. Psychol. 55, 162–170.
LaBar, K.S., Crupain, M.J., Voyvodic, J.T., McCarthy, G., 2003. Dynamic perception of facial affect and identity in the human brain. Cereb. Cortex 13, 1023–1033.
Labonte´, B., Suderman, M., Maussion, G., Navaro, L., Yerko, V., Mahar, I., et al., 2012. Genome-wide epigenetic regulation by early-life trauma. Arch. Gen. Psychiatry. 69, 722–731.
Loeber, R, Burke, J.D., Lahey, B.B., Winters, A., Zera, M., 2000. Oppositional defiant and conduct disorder: a review of the past 10 years, Part I. J. Am. Acad. Child Adolesc. Psychiatry 39, 1468–1484. https://doi.org/10.1097/00004583-200012000-00007.
Loveland, K.A., Pearson, D.A., Tunali-Kotoski, B., Ortegon, J., Gibbs, M.C., 2001. Judgments of social appropriateness by children and adolescents with autism. J. Autism Dev. Disord. 31, 367–376.
Manes, F., Sahakian, B., Clark, L., Rogers, R., Antoun, N., Aitken, M., Robbins, T., 2002. Decision-making processes following damage to the prefrontal cortex. Brain 125, 624–639.
Marsh, A.A., Blair, R.J.R., 2008. Deficits in facial affect recognition among antisocial populations: a meta-analysis. Neurosci. Biobehav. Rev. 32, 454–465. https://doi.org/10.1016/j.neubiorev.2007.08.003.
Murray, J., Farrington, D.P., 2010. Risk factors for conduct disorder and lelinquency: key findings from longitudinal studies. Can. J. Psychiatry 55, 633–642. https://doi.org/10.1177/070674371005501003.
Odgers, C.L., Moffitt, T.E., Broadbent, J.M., Dickson, N., Hancox, R.J., Harrington, H., Poulton, R., Sears, M.R., Thomson, W.M., Caspi, A., 2008. Female and male antisocial trajectories: from childhood origins to adult outcomes. Dev. Psychopathol. 20, 673–716. https://doi.org/10.1017/S0954579408000333.
Oldfield, R.C., 1971. The assessment and analysis of handedness: the Edinburgh inventory. Neuropsychologia 9, 97–113. https://doi.org/10.1016/0028-3932(71)90067-4.
Oostermeijer, S., Whittle, S., Suo, C., Allen, N.B., Simmons, J.G., Vijayakumar, N., et al., 2016. Trajectories of adolescent conduct problems in relation to cortical thickness development: a longitudinal MRI study. Transl. Psychiatry 6, e841. https://doi.org/10.1038/tp.2016.111.
Panizzon, M.S., Fennema-Notestine, C., Eyler, L.T., Jernigan, T.L., Prom-Wormley, E., Neale, M., et al., 2009. Distinct genetic influences on cortical surface area and cortical thickness. Cereb. Cortex 19, 2728–2735. https://doi.org/10.1093/cercor/bhp026.
Pujol, J., Harrison, B.J., Ortiz, H., Deus, J., Soriano-Mas, C., López-Solà, M., et al., 2009. Influence of the fusiform gyrus on amygdala response to emotional faces in the non-clinical range of social anxiety. Psychol. Med. 39, 1177–1187. https://doi.org/10.1017/S003329170800500X.
Raine, A., Yang, Y., 2006. Neural foundations to moral reasoning and antisocial behavior. Soc. Cognit. Affect. Neurosci. 1, 203–213. https://doi.org/10.1093/scan/nsl033.
Rakic, P., 2009. Evolution of the neocortex: a perspective from developmental biology. Nat. Rev. Neurosci. 10, 724–735. https://doi.org/10.1038/nrn2719.
Raznahan, A., Lerch, J.P., Lee, N., Greenstein, D., Wallace, G.L., Stockman, M., et al., 2011. Patterns of coordinated anatomical change in human cortical development: a

longitudinal neuroimaging study of maturational coupling. Neuron 72, 873–884. https://doi.org/10.1016/j.neuron.2011.09.028.
Rhee, S.H., Lahey, B.B., Waldman, I.D., 2015. Comorbidity Among Dimensions of Childhood Psychopathology: Converging Evidence from Behavior Genetics. Child Dev. Perspect. 9, 26–31. https://doi.org/10.1111/cdep.12102.
Rhee, S.H., Waldman, I.D., 2002. Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. Psychol. Bull. 128, 490–529.
Rogers, J.C., De Brito, S.A., 2016. Cortical and subcortical gray matter volume in youths with conduct problems: a meta-analysis. JAMA Psychiatry 73, 64–72. https://doi.org/10.1001/jamapsychiatry.2015.2423.
Sabatinelli, D., Fortune, E.E., Li, Q., Siddiqui, A., Krafft, C., Oliver, W.T., et al., 2011. Emotional perception: meta-analyses of face and natural scene processing. Neuroimage 54, 2524–2533. https://doi.org/10.1016/j.neuroimage.2010.10.011.
Salvatore, J.E., Dick, D.M., 2016. Genetic influences on conduct disorder. Neurosci. Biobehav. Rev. https://doi.org/10.1016/j.neubiorev.2016.06.034. In press.
Smaragdi, A., Cornwell, H., Toschi, N., Riccelli, R., Gonzalez-Madruga, K., Wells, A., et al., 2017. Sex differences in the relationship between conduct disorder and cortical structure in adolescents. J. Am. Acad. Child Adolesc. Psychiatry 56, 703–712. https://doi.org/10.1016/j.jaac.2017.05.015.
Sarkar, S., Daly, E., Feng, Y., Ecker, C., Craig, M.C., Harding, D., et al., 2015. Reduced cortical surface area in adolescents with conduct disorder. Eur. Child Adolesc. Psychiatry 24, 909–917. https://doi.org/10.1007/s00787-014-0639-3.
Saunders, J.B., Aasland, O.G., Babor, T.F., de la Fuente, J.R., Grant, M., 1993. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption–II. Addiction 88, 791–804.
Schiffer, B., Leygraf, N., Müller, B.W., Scherbaum, N., Forsting, M., Wiltfang, J., Gizewski, E.R., Hodgins, S., 2013. Structural brain alterations associated with schizophrenia preceded by conduct disorder: a common and distinct subtype of schizophrenia? Schizophr. Bull. 39, 1115–1128. https://doi.org/10.1093/schbul/sbs115.
Schwarz, K.A., Wieser, M.J., Gerdes, A.B.M., Mühlberger, A., Pauli, P., 2013. Why are you looking like that? How the context influences evaluation and processing of human faces. Soc. Cogn. Affect. Neurosci. 8, 438–445. https://doi.org/10.1093/scan/nss013.
Shannon, B.J., Raichle, M.E., Snyder, A.Z., Fair, D.A., Mills, K.L., Zhang, D., et al., 2011. Premotor functional connectivity predicts impulsivity in juvenile offenders. PNAS 108, 11241–11245. https://doi.org/10.1073/pnas.1108241108.
Sjöberg, R.L., Nilsson, K.W., Wargelius, H.L., Leppert, J., Lindström, L., Oreland, L., 2007. Adolescent girls and criminal activity: role of MAOA-LPR genotype and psychosocial factors. Am. J. Med. Genet. B Neuropsychiatr. Genet. 144B, 159–164. https://doi.org/10.1002/ajmg.b.30360.
Sowell, E.R., Peterson, B.S., Kan, E., Woods, R.P., Yoshii, J., Bansal, R., et al., 2007. Sex differences in cortical thickness mapped in 176 healthy individuals between 7 and 87 years of age. Cereb. Cortex 17, 1550–1560.
Steadman, H.J., Mulvey, E.P., Monahan, J., Robbins, P.C., Appelbaum, P.S., Grisso, T., Roth, L.H., Silver, E., 1998. Violence by people discharged from acute psychiatric inpatient facilities and by others in the same neighborhoods. Arch. Gen. Psychiatry 55, 393–401. https://doi.org/10.1001/archpsyc.55.5.393.
Straus, M.A., Hamby, L., S., Boney-McCoy, S., Sugarman, D.B., 1996. The Revised Conflict Tactics Scales (CTS2): development and preliminary psychometric data. J. Fam. Issues 17, 283–316. https://doi.org/10.1177/019251396017003001.
Wallace, G.L., White, S.F., Robustelli, B., Sinclair, S., Hwang, S., Martin, A., Blair, R.J.R., 2014. Cortical and subcortical abnormalities in youths with conduct disorder and elevated callous-unemotional traits. J. Am. Acad. Child Adolesc. Psychiatry 53, 456. https://doi.org/10.1016/j.jaac.2013.12.008. –65.e1.
Wechsler, D., 1991. The Wechsler Intelligence Scale for Children, 3rd ed. The Psychological Corporation, San Antonio, TX.
Wechsler, D., 1997. WAIS-III Administration and Scoring Manual. The Psychological Corporation, San Antonio, TX.
White, S.F., Williams, W.C., Brislin, S.J., Sinclair, S., Blair, K.S., Fowler, K.A., et al., 2012. Reduced activity within the dorsal endogenous orienting of attention network to fearful expressions in youth with disruptive behavior disorders and psychopathic traits. Dev. Psychopathol. 24, 1105–1116. https://doi.org/10.1017/S0954579412000560.
Winkler, A.M., Sabuncu, M.R., Yeo, B.T.T., Fischl, B., Greve, D.N., Kochunov, P., Nichols, T.E., Blangero, J., Glahn, D.C., 2012. Measuring and comparing brain cortical surface area and other areal quantities. Neuroimage 61, 1428–1443. https://doi.org/10.1016/j.neuroimage.2012.03.026.