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REDUCED CORTICAL THICKNESS AND INCREASED SURFACE AREA IN ANTISOCIAL PERSONALITY DISORDER

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Abstract—Antisocial personality disorder (ASPD), one of whose characteristics is high impulsivity, is of great interest in the field of brain structure and function. However, little is known about possible impairments in the cortical anatomy in ASPD, in terms of cortical thickness (CTh) and surface area (SA), as well as their possible relationship with impulsivity. In this neuroimaging study, we first investigated the changes of CTh and SA in ASPD patients, in comparison to those of healthy controls, and then performed correlation analyses between these measures and the ability of impulse control. We found that ASPD patients showed thinner cortex while larger SA in several specific brain regions, i.e., bilateral superior frontal gyrus (SFG), orbitofrontal and triangularis, insula cortex, precuneus, middle frontal gyrus (MFG), middle temporal gyrus (MTG), and left bank of superior temporal sulcus (STS). In addition, we also found that the ability of impulse control was positively correlated with CTh in the SFG, MFG, orbitofrontal cortex (OFC), pars triangularis, superior temporal gyrus (STG), and insula cortex. To our knowledge, this study is the first to reveal simultaneous changes in CTh and SA in ASPD, as well as their relationship with impulsivity. These cortical structural changes may introduce uncontrolled and callous behavioral characteristic in ASPD patients, and these potential biomarkers may be very helpful in understanding the pathomechanism of ASPD.

Key words: cortical anatomy, impulsivity, response inhibition, MRI, cortical thickness, surface area.

INTRODUCTION

Antisocial personality disorder (ASPD) describes patterns of antagonism and impulsively aggressive behaviors that begin in childhood and remain stable throughout the lifespan according to Diagnostic and Statistical Manual of Mental Disorders (DSM-V). Epidemiological studies report a prevalence of 2–3% in the general population, with estimates of approximately 3% in men and 1% in women (Gibbon et al., 2010). Furthermore, 47% of male prisoners in worldwide prison systems were diagnosed with ASPD (Fazel and Danesh, 2002), showing a very close link between ASPD and criminal behavior.

Abnormal brain structures are found to correlate with ASPD, based on morphological MRI studies. Raine et al. found that the prefrontal gray matter volume in ASPD was reduced by about 11% in comparison to that of the control group (Raine et al., 2000). The reduced gray matter volume in the prefrontal cortex was replicated in many other studies of ASPD (Yang and Raine, 2009). Reduced gray matter volume of temporal regions was also revealed in ASPD (Bassarath, 2001; Barkataki et al., 2006). Additionally, the relationship between impulsive aggression and the reduced volume of the frontal lobe was also found in ASPD patients (Raine et al., 2000; Laakso et al., 2002). These studies of ASPD mainly focused on gray matter volume. However, the cortical gray mater volume is essentially jointly determined by biologically distinct cortical attributes of cortical thickness (CTh) and surface area (SA), each having its own cellular mechanism and genetic underpinning, thus providing unique and complementary information of the cortex (Chen et al., 2013; Lyall et al., 2015; Li et al., 2016). More importantly, CTh and SA have been found distinctively correlated with cognitive functions, but also differentially affected in various brain disorders (Lyall et al., 2015). Hence, studying CTh and SA can better capture subtle, but important, cortical changes associated with ASPD. Ly et al. found that psychopathy patients showed cortical thinning in a number of regions, specially the left insula, bilateral anterior temporal cortices and right inferior frontal

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Abbreviations: ACC, anterior cingulate cortex; ASPD, antisocial personality disorder; CTh, cortical thickness; DSM-V, diagnostic and statistical manual of mental disorders; EF, executive function; GLM, general linear model; MFG, middle frontal gyrus; OFC, orbitofrontal cortex; SA, surface area; SFG, superior frontal gyrus; SFS, superior temporal sulcus; STG, superior temporal gyrus.

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gyrus (Ly et al., 2012). Though psychopathy shares behavioral overlap with the clinical diagnosis of ASPD, ASPD is not synonymous of psychopathy (Blair, 2012). Currently, the study of cortical anatomy on ASPD population is still scarce. The changes in CTh and SA in ASPD still remain unclear.

Impulsivity is a central and important characteristic of ASPD according to DSM-V. Response inhibition, i.e., impulse control is the main impulsivity variable (Dougherty et al., 2005). Swann et al. found that ASPD was characterized more by increased rapid-response impulsivity, while aspects of impulsivity about reward-delay or attention appear relatively intact (Swann et al., 2009). GoStop impulsivity paradigm developed by NRLC-group can measure the level of impulse control or rapid-response impulsivity precisely (Dougherty et al., 2005). This paradigm has been used in all kinds of people, e.g., borderline personality disorder (BPD) (Cackowski et al., 2014), internet addiction (Li et al., 2014a), and substance abuse (Coffey et al., 2011). But to date, there is no study on the correlation between CTh/SA changes and the levels of impulse control in ASPD.

In this study, we hypothesized that there were distinct alterations in CTh and SA in ASPD, and there also were potential relationships between these structural changes and the ability of impulse control. This study will provide valuable information regarding to the abnormal neuroanatomy of ASPD, while also highlighting the potential relations between structural changes and impulsivity in ASPD patients.

EXPERIMENTAL PROCEDURES

Participants

The School for Youth Offender of Hunan Province performed ‘‘Enclosed-style” management and reformatory education for those offenders under 18 years of age, when committing certain crimes, e.g., robbery and violent attacks. We recruited volunteers at legal age (18 years of age at scan) from this school. The participants were diagnosed whether with ASPD or not, according to the following steps. In the first step, all the volunteers in groups were tested by a professional using the Personality Diagnostic Questionaire-4 +(PDQ-4 +). For those ASPD scores equal to or above 4 score, two senior psychiatrists then tested whether they had Axis 1 disorders of major mental illness and excluded those with Axis 1 disorders. The remaining volunteers were continuously tested using the structured clinical interview for DSM-IV (SCID-II) by the same psychiatrists. The SCID-II is a diagnostic exam to determine personality disorders. Finally, 27 subjects were diagnosed with only ASPD, i.e., all ASPD disorders met both PDQ-4 criteria and SCID-II criteria for ASPD. We also chose 25 healthy control subjects, who met neither PDQ-4 criteria nor SCID-II criteria for ASPD. All the controls were tested using the same methods as ASPD disorders by the same psychiatrists. The control subjects were matched to the ASPD subjects in age, education, and IQ (Table 1). IQ score of each subject was obtained using Wechsler Adult Intelligence Scale. One-way ANOVAs were performed on the demographics of the groups to test whether the groups were well matched.

Table 1. Characteristics of the participants in this study

	ASPD (Mean ± SD)	Controls (Mean ± SD)	*p*-Value
Number	27	25	–
Gender	27 males	25 males	–
Age (Years)	20.30 ± 3.01	21.13 ± 3.16	0.385
Education (Years)	9.54 ± 3.42	10.26 ± 2.33	0.753
IQ	94.55 ± 8.62	95.30 ± 10.5	0.797

ASPD: Offenders with antisocial personality disorder.

All subjects were right-handed native Chinese speakers, with no history or current diagnosis of drugs abuse, and they were kept away from alcohol at least 6 months before the experiment. They were accompanied by three instructors individually during the experiment. This study was approved by the Ethics Committee of the Third Xiangya Hospital of Central South University and also the Ethics Committee of the School for Youth Offender of Hunan Province. All volunteers signed on the written informed consent after they understood the study.

Impulsivity measure

In the task, we used GoStop impulsivity paradigm to measure the level of impulse control. Black five-digit numbers are presented successively on the computer screen (with each 500 ms following by 1-s blank screen). When the present number is the same as the previous number, the participant should press a mouse button as quickly as possible (‘no-stop trials’). When the target’s color changes from black to red (‘stop signal’, occurring from 50, 150, 250 to 350 ms after stimulus onset), the response needs to be withheld (‘stop trials’). If the five-digit number does not exactly match the previous number, the participant must withhold responding (‘novel trials’). In our study, no-stop trials were 25% of the trials, stop trials 25%, and novel trials 50%. The total number of all trails was 160 and the task lasted 240 s. Before the normal task, the participants received a detailed description of the task and underwent a training session of 60 s so as to be familiar with the task. To ensure cooperation, the response accuracy of each subject during ‘‘novel trials” and ‘‘stop trials” were calculated. The subjects whose response accuracies in the ‘‘novel trials” and ‘‘stop trials” conditions were lower than 80% were excluded. The level of impulse control is calculated as the percentage of successful inhibition. Lower percentages of successful inhibitions (during ‘stop trials’) indicate more difficulties with response inhibition.

To investigate whether there were significant differences between groups under different delay tasks, independent-sample *t*-tests were employed.

Image acquisition

T1-weighted structural magnetic resonance images were acquired at the Third Xiangya Hospital of Central South

University on a 3T Philips scanner. The principal sequence of this study was an ultrafast gradient echo 3D sequence (T1W_3D_TFE_ref). Scans were acquired in the sagittal plane with the following parameters: TR = 7.4 ms, TE = 3.5 ms, TI = 900 ms, gap = 0.6 m m, flip angle = 8°, BW = 210 Hz/ pixel, FOV = 228 × 2 27 mm, matrix = 240 × 240; 301 slices with resolution = 1.04 * 1.04 * 0.6 mm^3, acquisition time = 4 min 58 s. Images were inspected for motion artifact at the time of acquisition and scans were repeated as necessary. Images were also reviewed and discarded, if there were any pathological findings.

Image processing

All MR images were processed on the same workstation using FreeSurfer version 5.3 (http://surfer.nmr.mgh.harvard.edu/). FreeSurfer morphometric procedures have good test–retest reliability across different scanner manufacturers or field strengths (Cannon et al., 2015), and measuring methods of CTh and SA have also been extensively validated in normal subjects or patients with various brain disorders (McLaughlin et al., 2014; Zielinski et al., 2014; Cannon et al., 2015).

CTh and SAs measures at each vertex were calculated based on cortical surface reconstruction (Fischl et al., 2004). First, several important processing steps were performed, including skull stripping, spatial transformation, and atlas registration. Then, both gray matter/white matter and gray matter/cerebrospinal fluid (CSF) surfaces were reconstructed and parcellated into different regions based on the folding structures of gyri and sulci (Desikan et al., 2006). CTh was calculated as the shortest distance between the gray/white surface and the pial surface (Fischl and Dale, 2000). More technical details of these procedures were described previously (Fischl et al., 2004; Desikan et al., 2006). The results of the automated segmentation, surface reconstruction, and parcellation process were manually inspected for all subjects, with no abnormality found.

Statistical analyses

As a first step, CTh and SA maps were smoothed using a 20-mm Gaussian kernel (Karama et al., 2014). We also smoothed the map using other three Gaussian kernels, i.e., 10 mm, 15 mm and 25 mm and found there were similar results. So we would only report the results smoothed using the 20-mm Gaussian kernel. Then, a general linear model (GLM) implemented in Freesurfer was used to investigate whether there were significant differences in measures of CTh and SA at each vertex (separately) between ASPD patients and control subjects. The false-discovery rate (FDR) of 0.05, corrected for multiple comparisons at the vertex level, was used in this study. We also modeled the age, IQ, and whole brain volume as covariates in order to minimize any confounding effects of these variables on the findings, i.e., parameter estimates for y_{ij}(CTh/SA) and the main effect of group G_j were estimated by regression of a GLM at each vertex i and subject j, with the age, IQ, and total brain volume as covariates:

$$y_{ij} = \beta_0 + \beta_1 G_j + \beta_2 Age_j + \beta_3 IQ_j + \beta_4 Vol_j + \varepsilon_{ij}$$

where ε is the residual error.

Secondary analyses were conducted to determine the correlation of the ability of impulse control, i.e., the percentage of successful inhibition during different task, and the regional measures showing evidence of difference between two groups. First, each cluster identified at the first step was extracted. Second, average CTh and total SA in each cluster were calculated for each subject. Finally, the Pearson correlation coefficient was calculated between the percentage of successful inhibition and average CTh *or* total SA of each cluster ($P < 0.05$, uncorrected).

RESULTS

Behavioral performance

The ASPD patients showed impaired response inhibition during the Go-Stop task (Fig. 1 and Table 2). The percentage of successfully inhibited responses was significantly lower in the ASPD patients, compared with the controls, during a 50-ms, 150-ms and 250-ms delay task ($p < 0.05$). When the task became harder (from 50-ms delay to 150-ms delay, to 250-ms delay, and to 350-ms delay), both groups demonstrated more and more difficulty in withholding their responses, as their percentage of successful inhibition was lower and lower. However, it was observed that the ASPD patients had more difficulties in response inhibition compared to healthy controls under the same task. Although the differences did not reach significance ($p > 0.05$) during the 350-ms delay tasks, the control group noticeably performed better.

Reduced CTh in ASPD patients

On the left hemisphere, statistical analyses revealed seven clusters ($p < 0.05$, FDR corrected) that differed significantly in thickness between ASPD patients and healthy controls (Fig. 2 and Table 3). Importantly, all the seven clusters had reduced thickness in ASPD patients, compared with healthy controls. Specifically, they were located in the superior frontal gyrus (SFG)/rostral anterior cingulate cortex (ACC), precuneus, pars orbitalis/orbitofrontal cortex (OFC), pars triangularis, insula cortex, superior temporal sulcus (SFS) and superior temporal gyrus (STG). The average CTh in each of the seven clusters was calculated for each subject.

On the right hemisphere, statistical analyses revealed six clusters ($p < 0.05$, FDR corrected) that differed significantly between the two groups, with ASPD exhibiting reduced CTh in all clusters, when compared with control subjects (Fig. 2 and Table 3). These six clusters were located at the SFG/rostral ACC, precuneus, caudal middle frontal gyrus (MFG), pars orbitalis/OFC, pars triangularis and insula/pars triangularis/OFC. Similarly, the average CTh in each of the 6 clusters was calculated for each subject.

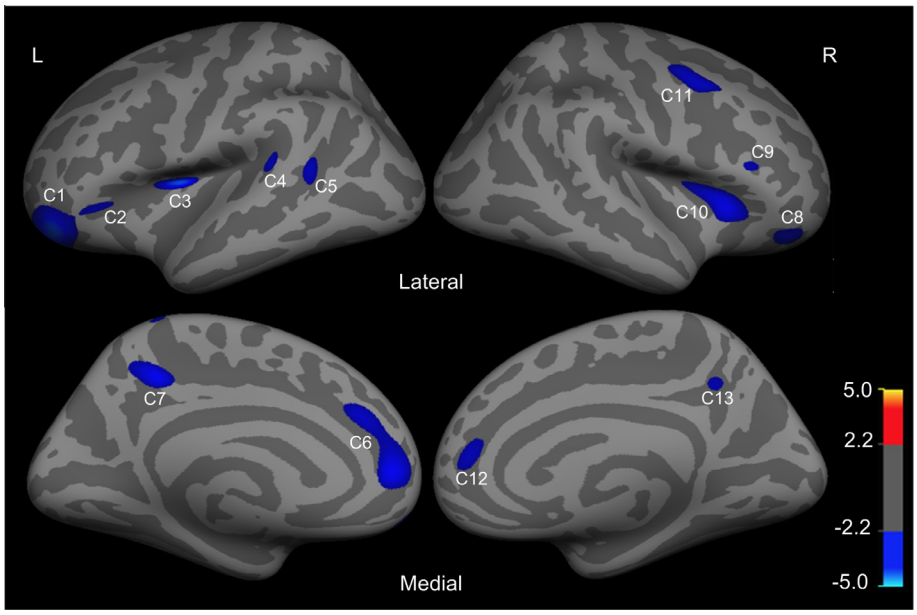

Fig. 1. Regions in the left hemisphere (L) and right hemisphere (R) with significant reductions in cortical thickness (CTh) in ASPD patients relative to control subjects (false discovery rate correction (FDR), $p < 0.05$). The color bar indicates t value.

Table 2. The behavioral performance of ASPD patients and healthy controls (HC) during the Go-Stop task

Delay (ms)	ASPD (Mean ± SD)	HC (Mean ± SD)	*t*-Value	*p*-Value
50	80.0% ± 20%	93.5% ± 16%	2.382	0.021
150	62.5% ± 30%	83.8% ± 16%	3.153	0.003
250	43.2% ± 29%	70.0% ± 28%	3.420	0.001
350	30.0% ± 25%	45.4% ± 34%	1.386	0.072

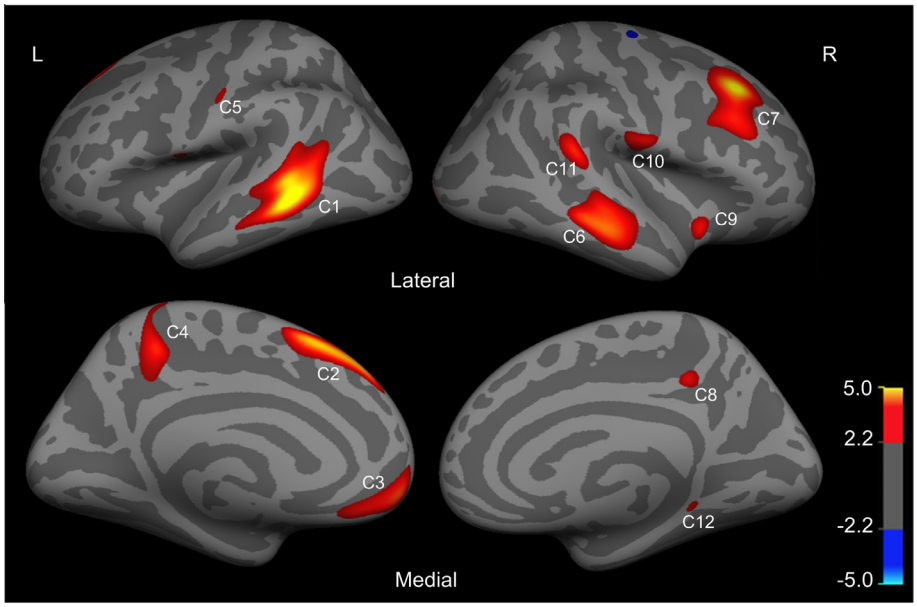

Fig. 2. Regions in left hemisphere (L) and right hemisphere (R) with significant increase in surface area (SA) in ASPD patients relative to control subjects (false discovery rate correction (FDR), $p < 0.05$). The color bar indicates t value.

Table 3. Significant alterations in cortical thickness and surface area in ASPD patients relative to control subjects

Cluster	Hemisphere	Brain region	Peak vertex Talairach			*t* value	*p* value (FDR corrected)	Size (mm^2)	Percentage of change
			x	*y*	*z*				
Cortical thickness (mm)									
C1	Left	Orbitofrontal/pars orbitalis	−29.8	44.2	−12.0	−3.674	0.000	1483.31	−8.30
C2	Left	Pars triangularis	−38.7	31.7	−2.9	−2.429	0.018	108.34	−7.43
C3	Left	Insula/pars triangularis	−34.2	−2.2	14.0	−3.811	0.000	194.99	−6.60
C4	Left	Bank of superior temporal sulcus	−47.7	−45.5	13.2	−2.533	0.014	139.65	−3.70
C5	Left	Superior temporal gyrus	−50.4	−37.5	13.8	−2.553	0.013	104.75	−7.09
C6	Left	Superior frontal gyrus/rostral anterior cingulate cortex	−11.1	43.9	2.7	−2.989	0.004	650.26	−7.75
C7	Left	Precuneus	−7.9	−40.5	41.5	−2.995	0.004	290.19	−6.25
C8	Right	Orbitofrontal/pars orbitalis	30.2	38.1	−10.1	−2.636	0.011	265.12	−6.14
C9	Right	Pars triangularis	48.1	26.1	13.2	−2.379	0.021	60.04	−9.29
C10	Right	Insula/pars triangularis/ orbitofrontal	29.5	23.1	5.3	−3.069	0.003	433.06	−7.59
C11	Right	Caudal middle frontal gyrus	39.8	6.7	6.5	−2.761	0.008	543.18	−9.89
C12	Right	Superior frontal gyrus/rostral anterior cingulate cortex	15.5	41.2	7.9	−2.508	0.015	110.92	−9.09
C13	Right	Precuneus	14.1	−47.3	38.5	−2.426	0.018	49.34	−6.58
Surface area (mm^2)									
C1	Left	Middle temporal gyrus/superior temporal sulcus	−61.4	−42.8	−0.7	5.645	0.000	2227.48	10.70
C2	Left	Superior frontal gyrus	−7.0	22.3	49.7	4.665	0.000	1075.83	5.11
C3	Left	Medial orbitofrontal	−8.3	53.6	−10.3	3.783	0.000	816.17	6.79
C4	Left	Precuneus	−12.4	−41.7	46.4	3.534	0.001	501.69	9.22
C5	Left	Postcentral gyrus	−54.0	−13.5	36.8	2.751	0.008	79.24	8.70
C6	Right	Middle temporal/superior_temporal	59.0	−23.9	−10.2	4.058	0.000	1570.22	8.44
C7	Right	Middle frontal/superior frontal	30.2	26.2	38.2	5.206	0.000	1728.3	6.25
C8	Right	Precuneus	6.9	−38.0	40.8	3.038	0.004	89.47	9.89
C9	Right	Insula	36.4	6.2	−7.4	3.373	0.000	100.19	3.23
C10	Right	Precentral	62.5	−10.8	16.3	3.244	0.002	262.28	2.49
C11	Right	Supramarginal	20.8	−96.6	−1.1	2.669	0.010	145.46	9.94
C12	Right	Parahippocampal	18.2	−35.7	−6.9	2.825	0.007	30.03	9.80

Increased SA in ASPD patients

On the left hemisphere, statistical analyses revealed five clusters ($p < 0.05$, FDR corrected) that differed significantly in SA between ASPD patients and healthy controls (Fig. 3 and Table 3). Interestingly, all these 5 clusters had increased SA in ASPD patients compared with healthy controls. Specifically, they were located in the STG/bank of SFS, SFG, medial OFC, precuneus and postcentral gyrus. The total SA of each cluster was calculated for each subject.

On the right hemisphere, statistical analyses revealed seven clusters ($p < 0.05$, FDR corrected) that differed significantly between the two groups, with ASPD exhibiting increased SA compared with control subjects in all clusters (Fig. 3 and Table 3). These seven clusters were located at the middle temporal/STG, SFG, MFG, precuneus, insula cortex, precentral gyrus, supramarginal gyrus and parahippocampal gyrus. The total SA of each of the seven clusters was calculated for each subject.

Correlation between cortical measures and the ability of impulse control

For average CTh in each cluster, we performed correlation analysis with the percentage of successful inhibition. During 50-ms delay, CTh was correlated positively and significantly with the percentage of successful inhibition in the following clusters ($P < 0.05$, uncorrected): C3 (left insula cortex, Fig. 3A), C5 (left STG, Fig. 3B), C6 (left SFG, Fig. 3C), C10 (right insula cortex, Fig. 3D), and C11 (right caudal middle frontal cortex, Fig. 3E). During 150 ms delay, CTh was correlated positively and significantly with the percentage of successful inhibition in the following clusters ($P < 0.05$, uncorrected): C3 (left insula cortex, Fig. 4A), C4 (left bank of STG, Fig. 4B), C5 (left STG, Fig. 4C), C6 (left SFG, Fig. 4D), C9 (right pars triangularis, Fig. 4E), C10 (right insula cortex, Fig. 4F), C11 (right caudal MFG, Fig. 4G), and C12 (right SFG, Fig. 4H). During 250-ms delay, CTh in the C9 (right pars triangularis, Fig. 5A) and C10 (right insula, Fig. 5B) was correlated positively and significantly with the percentage of successful inhibition ($P < 0.05$, uncorrected). In other clusters, there existed also positive correlational trends, although not significant. These results demonstrate that the ability of impulse control is, in fact, correlated with CTh, i.e., response inhibition is more difficult, if CTh is thinner.

We also performed correlation analysis between the percentage of successful inhibition and the SA of each cluster, but no significant results were obtained.

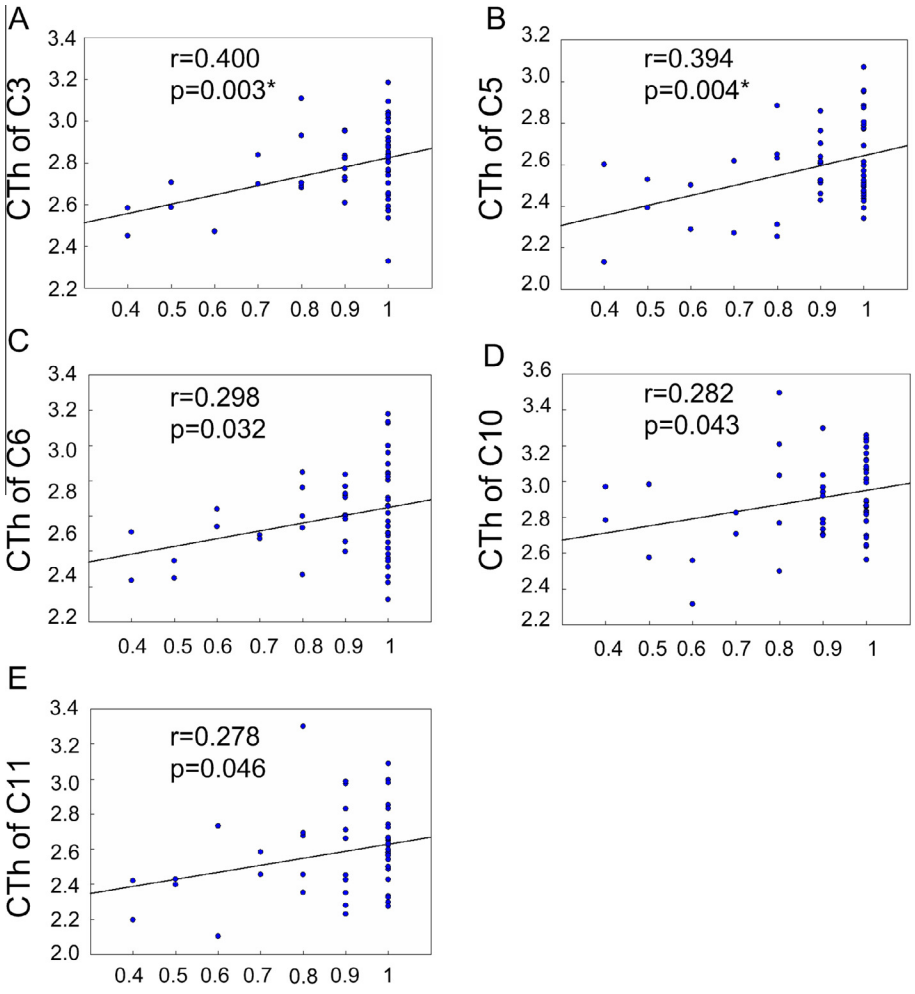

Fig. 3. Significant correlation between behavioral performance and cortical thickness (CTh, mm) in clusters under 50 ms delay task. The horizontal axis shows the percentage of successfully inhibited response. C denotes Cluster. $^{*}p < 0.05$ under false discovery rate correction.

DISCUSSION

In this study, we investigated the ability of impulse control in ASPD patients, the changes of CTh and SA in ASPD patients, and the relationship between CTh/SA and impulsivity. We found that the percentage of successfully inhibited responses was significantly lower in ASPD patients, which suggests impaired response inhibition in ASPD patients. Though this result is in line with DSM-V and previous studies that had reported poor impulse control in ASPD patients (Verona et al., 2012; Swann et al., 2013), it well quantified the ability of response inhibition. A more important finding is that ASPD patients present reduced CTh, but increased SA, when compared with healthy controls, while the traditional cortical volume could obscure information about CTh and SA, which are the two biologically distinct determinants of cortical volume (Raznahan et al., 2011).

Abnormalities in the prefrontal cortex (PFC) have been widely found in many previous imaging studies of ASPD (Yang and Raine, 2009; Raine et al., 2011). PFC is believed to be responsible for executive functions (EFs) of ASPD (Morgan and Lilienfeld, 2000; Dolan, 2002), and neuropsychological deficits in EF may introduce severe antisocial and aggressive behavior (Ogilvie et al., 2011). A damage study found that the injured PFC resulted in an increase in impulsivity, a decrease of judgment ability, and a lack of decision-making ability (Damasio et al., 1994). Specially, the SFG is believed to be involved in self-awareness (Goldberg et al., 2006) and acted as functional operators when loading, maintaining, and switching between distinct stimulus–response tasks (Cutini et al., 2008). Abnormalities of SFG may be associated with disturbances of executive functioning, cognition, and judgment (Loveland et al., 2001). Rajah et al. also suggested that the MFG might mediate response selection and monitoring processes (Rajah et al., 2008).

Notably, the OFC extending to pars orbitalis and pars triangularis showed significantly reduced CTh in ASPD in our results. Our prior study also found that there were abnormal functional connections within OFC in ASPD patients (Tang et al., 2013). Major functions ascribed to OFC are the regulation of emotional responses, especially under threatening or risky situations (Buchheim et al., 2013) and behavioral inhibition (Christopoulos et al., 2009; Lopez-Caneda et al., 2012). The lateral OFC plays an important role in conflict resolution and

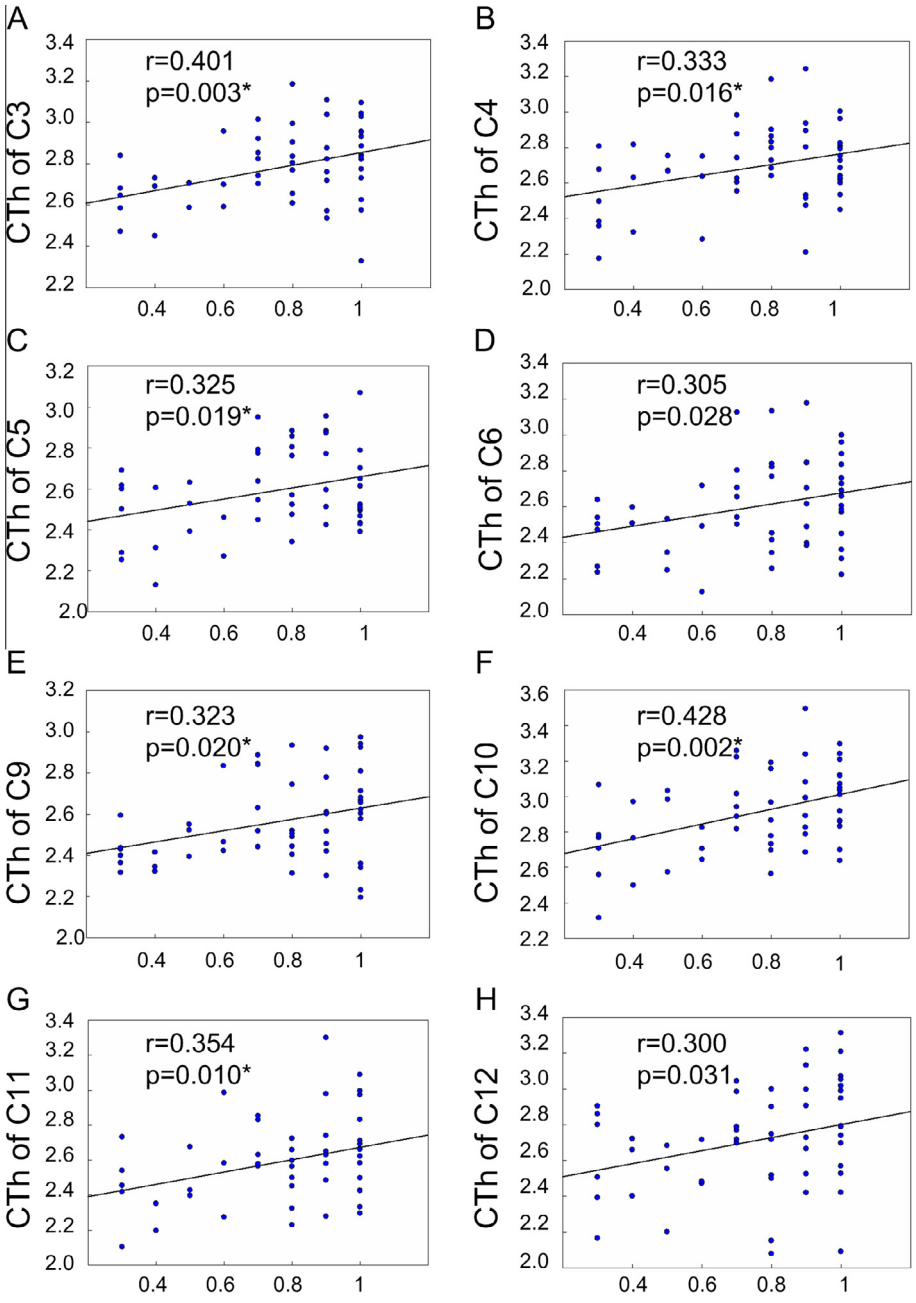

Fig. 4. Significant correlation between behavioral performance and cortical thickness (CTh, mm) in clusters under 150 ms delay task. The horizontal axis shows the percentage of successfully inhibited response under different delay tasks. C denotes Cluster. $^{*}p < 0.05$ under false discovery rate correction.

encodes new expectations about punishment and social reprisal (Campbell-Meiklejohn et al., 2012). The mesial PFC is highly involved in emotional processing and is more regularly activated by emotional tasks than by cognitive tasks (Steele and Lawrie, 2004). OFC injures may lead to severe antisocial behaviors with the symptoms of antisocial disorders (Seguin, 2004). Disruption of this area's activity by transcranial magnetic stimulation results in changes in risk attitudes in decision-making behaviors (Fecteau et al., 2007). Individuals with OFC lesions generally presented certain behavioral patterns of social inappropriate, misinterpreting others' moods, impulsivity and risk (Tranel et al., 2002; Seguin, 2004).

Our evidence of thinner CTh and increased SA in PFC, amalgamated those prior converging lines of evidence, further implicates that prefrontal regions contribute to the neuropathology of ASPD. If PFC cannot function properly, a person may act impulsively and inappropriately, producing antisocial emotion. Hence, the changes of CTh and SA in PFC are significantly compromised and may serve as structural biomarkers for ASPD.

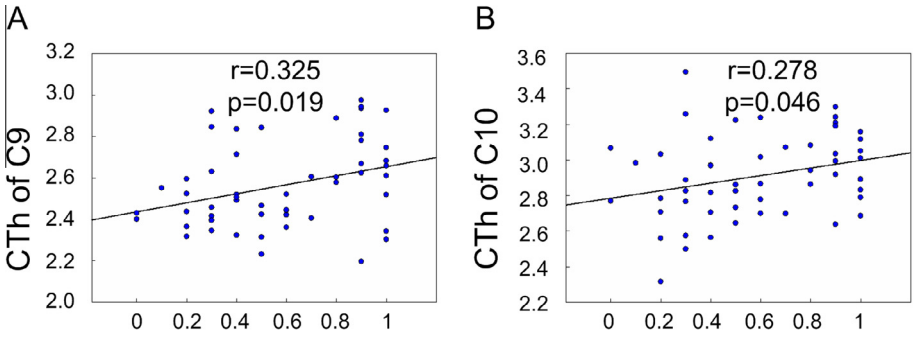

Fig. 5. Significant correlation between behavioral performance and cortical thickness (CTh, mm) in clusters under 250 ms delay task. The horizontal axis shows the percentage of successfully inhibited response. C denotes Cluster.

In our research, the left STG showed reduced CTh, while bilateral middle temporal gyrus (MTG) and STG showed increased SA. Jastorff et al. found that the main cognitive function of MTG was to evaluate action rationality (Jastorff et al., 2011). The temporal lobe takes part in sensory, affective, and higher cognitive processing (Kolb and Whishaw, 1990), and is also involved in language comprehension and emotion association (Arfken, 2009). The impairment in emotional comprehension and contagion may introduce changes in comportment and social behavior (Zahn et al., 2009; Rascovsky et al., 2011).

The abnormal temporal and frontal lobes occurred together in ASPD offenders in this study, which may increase the risk of anger and aggression, compared with each area independently (Potegal, 2012). Frontal–temporal joint dysfunction is believed to predispose to a patient's antisocial behavior (Miller et al., 1997), and the frontal–temporal hypoactivity is suggested to be a trait of severe violent crimes (Anckarsater et al., 2007). Psychopathy has multiple overlaps with ASPD in diagnostic criteria. Prior studies demonstrated co-occurring lesions in frontal-temporal regions in psychopathy (de Oliveira-Souza et al., 2008; Muller et al., 2008). Especially, psychopaths were also found to have thinner cortex in the left insula, bilateral anterior temporal cortices and right inferior frontal gyrus, compared to healthy controls (Ly et al., 2012).

In our study, bilateral anterior insula cortex and precuneus represented significantly reduced CT but increased SA in ASPD. The anterior insular cortex is believed to be responsible for emotional feelings, including anger, fear, sadness, social exclusion, and empathy (Craig, 2009; Vilares et al., 2012), and be important for motor impulsivity and reactive aggression (Dambacher et al., 2015). Especially, it is believed to be involved in the processing of norm violations (Sanfey et al., 2003). As for the precuneus, it plays an essential role in conscious information processing, specifically involved in self-reflection processes (Cavanna and Trimble, 2006; Cavanna, 2007). In brief, the functions of the regions (i.e., PFC, MTG/STG, Insula and Precuneus) in our study are important for selection, control and performance of socially relevant behavior. Injury to these cortical regions may cause violent and aggressive behavior, and cause a feature of callousness in ASPD.

Interestingly, these main brain regions with cortical thinning in ASPD offenders were also found in subjects with Conduct Disorder (CD). ASPD individuals have evident conduct problems by the age of 15 according to DSM-5′s criteria for ASPD, and about 25–40% of youths with conduct disorder will develop into ASPD in adulthood (Zoccolillo et al., 1992). Fahim et al. observed reduced CTh in STG, insula, and OFC in children with CD (Fahim et al., 2011). The thinner STG was also found in CD adolescents and showed a negative correlation with callous-unemotional traits (Wallace et al., 2014). More recently, male youths with CD showed abnormal cortical measurements in the STG, OFC and the insula relative to healthy controls (Fairchild et al., 2015). A meta-analysis study had also demonstrated gray matter reductions in youths with conduct problems, mainly within the insula, amygdala, frontal and temporal regions (Rogers and De Brito, 2016). So the thinner cortex in some brain regions in ASPD may be the sustainment from their childhood to adolescent then to adult.

The CTh and SA reflect different cellular mechanisms, i.e., CTh is mainly determined by the horizontal layers in the cortical columns including neurons and neuropil, whereas SA reflects the number of radial columns perpendicular to the pial surface (Rakic, 2009; Raznahan et al., 2011). In our study, the ASPD showed decreased CTh but increased SA, which may imply that ASPD subjects had less horizontal layers in the cortical columns but larger number of radial columns. The increased SA in the current study may occur due to the reduced CTh and result in ''inefficient'' or compensatory of cognitive control functions.

Finally, our study, for the first time, found the ability of impulse control is correlated with CTh in the SFG, MFG, STG, pars orbitofrontal and triangularis, and insula cortex. From the discussion above, we know that these regions are correlated with impulsivity. Response inhibition may be more difficult when CTh in these regions is thinner. Of note, the relationship between impulsive aggression and the reduced volume of the frontal lobe was also found in ASPD patients (Raine et al., 2000; Laakso et al., 2002). SA didn't show significant correlation with impulse control, which could imply that impulse control may not be strongly affected by SA.

The trials under different conditions (the 50/150/250/350-ms delay) were limited, and we will add

more trails to our studies in the future. In our study, though the SA showed no significant correlation with impulse control, it may be correlated with other features of ASPD, which will be investigated in future. In addition, our subject number should be increased. Currently, our subjects are all males and thus these findings cannot be directly generalized to the female subjects. Also, our correlation finding should be considered exploratory.

To our knowledge, this study is the first to reveal simultaneous changes of CTh and SA in ASPD, as well as the relationship between these structural changes and impulsivity. These specific indicators in specific brain regions could provide structural biomarkers for ASPD and potentially be helpful for understanding the pathomechanism of ASPD.

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