



Abnormal cortical gyrification in criminal psychopathy

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ABSTRACT

Background: Psychopathy is a personality disorder characterized by interpersonal and emotional abnormalities (e.g., lack of empathy and guilt) and antisocial behavior. Psychopathy has been associated with a number of structural brain abnormalities, most notably in orbital frontal and anterior/medial temporal regions, that may underlie psychopathic individuals' problematic behaviors. Past research evaluating cortical structure in psychopathy has considered thickness and volume, but to date no study has investigated differences in cortical gyrification, a measure of cortical complexity thought to reflect early neurodevelopmental cortical connectivity. **Methods:** We measured the local gyrification index (LGI) in a sample of 716 adult male inmates and performed a whole brain analysis assessing the relationship between LGI and total and factor scores on the Hare Psychopathy Checklist-Revised (PCL-R).

Results: PCL-R scores were negatively associated with LGI measures within the right hemisphere in the mid-cingulate cortex (MCC) and adjacent regions of the superior frontal gyrus as well as lateral superior parietal cortex. Additionally, PCL-R Factor 1 scores (interpersonal/affective traits) predicted less LGI within the right MCC and adjacent dorsomedial frontal cortex and greater LGI in bilateral occipital cortex. Scores on PCL-R Factor 2, indicating impulsivity and antisocial behaviors, did not predict LGI in any regions.

Conclusions: These findings suggest that psychopathy, particularly the interpersonal and affective traits, are associated with specific structural abnormalities that form during neurodevelopment and these abnormalities may underlie aberrant brain functioning in regions important in emotional processing and cognitive control.

Psychopathy is a personality disorder characterized by interpersonal and emotional abnormalities, such as superficial charm, callousness, and lack of guilt and remorse (Cleckley, 1976). These failures of socialization come at a high cost to the individual and to society. Despite a prevalence of approximately 1% in the general population, psychopathic individuals comprise approximately 20% of the prison population (Hare, 1996). They commit two to three times more violent and non-violent crimes than non-psychopathic offenders and recidivate at a much higher rate (Hare, 2003). Thus, psychopathic individuals contribute disproportionately to the estimated \$3.2 trillion annual cost of

crime in the United States (Anderson, 2012). Identifying the neural substrates that underlie the core deficits associated with psychopathy would shed light on these problematic behaviors.

Research has demonstrated that psychopathy is associated with a number of affective and cognitive abnormalities, which may underlie some of their core behavioral problems. Notably, psychopathic individuals have impairments in the way they process and respond to affective information (Baskin-Sommers and Newman, 2013; Blair et al., 2005; Blair et al., 1997; Book et al., 2007; Kiehl et al., 1999; Patrick, 2007). In addition to emotional processing abnormalities, psychopathic

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individuals demonstrate deficits in various cognitive processes, such as selective attention (Hiatt et al., 2004; Newman and Baskin-Sommers, 2011; Zeier et al., 2009), response reversal (Budhani et al., 2006), and inhibition (Brazil et al., 2012; Kiehl et al., 2000; Lapierre et al., 1995). Together, these affective and cognitive deficits result in the difficulties psychopathic individuals have in regulating their behavior and their callous attitudes towards others.

Consistent with these abnormalities, prominent neurobiological models of psychopathy have highlighted dysfunction in subcortical and fronto-temporal cortical areas involved in both emotion regulation and cognitive-behavioral control as the basis for the emotional and self-regulatory deficiencies seen in these individuals (Blair, 2007; Kiehl, 2006; Raine and Yang, 2006). Structural brain findings support these models, with abnormalities observed in volume or cortical thickness in the hippocampus, amygdala, prefrontal cortex, insula, temporal lobes, striatum and the anterior cingulate (Baskin-Sommers et al., 2016; de Oliveira-Souza et al., 2008; Ermer et al., 2012; Glenn, Raine, et al., 2010; Ly et al., 2012; Müller et al., 2008; Raine et al., 2004; Yang et al., 2009). Collectively, structural abnormalities in these regions are consistent with a prominent theory positing that psychopathy is characteristic of dysfunction in the broader paralimbic network (Ermer et al., 2012; Kiehl, 2006). Ly et al. (2012) further demonstrated that cortical thinning in portions of this network was associated with decreased functional connectivity within these regions, indicating that structural abnormalities may adversely impact function in areas implicated in psychopathic behaviors.

The vast majority of these previous studies examine structure through measurements of cortical volume and surface. However, one structural measure that has not yet been studied and may be of use in characterizing abnormalities in cortico-connectivity is cortical folding, or gyrification. Cortical gyrification represents the density and depth of sulci or the amount of buried cortical surface compared to visible cortex (Schaer et al., 2008; Zilles et al., 1988). Thus, gyrification is distinct from other measurements of the cortex such as surface area and thickness. The process of brain gyrification is thought to happen primarily before birth in the second and third trimester (Chi et al., 1977) and serves to optimize neural connectivity through compact wiring (Im et al., 2008; Van Essen, 1997; White et al., 2010). The ratio between the coronal perimeter with and without sulci, or the gyrification index (GI; Zilles et al., 1988), was initially thought to remain constant throughout childhood and into adulthood (Armstrong et al., 1995). However, recent research suggests there may be some changes continuing through adolescence (Alemán-Gómez et al., 2013; Klein et al., 2014; White et al., 2010) and adulthood (Hogstrom et al., 2013) as the brain undergoes continued developmental processes such as synaptic pruning (Gogtay et al., 2004; Lenroot and Giedd, 2006; White et al., 2010). Aberrations in cortical folding have been demonstrated in a number of clinical disorders, including Williams syndrome (Kippenhan et al., 2005; Schmitt et al., 2002), autism (Hardan et al., 2004; Wallace et al., 2013), schizophrenia (Palaniyappan et al., 2011; Vogeley et al., 2000; White et al., 2003) as well as personality traits linked to psychopathology (Miskovich et al., 2016), and have been linked to abnormal structural (Schaer et al., 2013) and functional (Dauvermann et al., 2012; Nixon et al., 2014) connectivity. In children, differences in cortical folding have been linked to conduct disorder and callous-uneemotional traits (Fairchild et al., 2015; Hyatt et al., 2012; Smaragdi et al., 2017). Since some prominent antisocial characteristics, particularly the affective and interpersonal dimensions of psychopathy, are associated with chronic antisocial behaviors across development into adulthood (Loeber et al., 2009), cortical folding abnormalities may be associated with vulnerability to psychopathology. However, no study has investigated cortical gyrification in psychopathy.

In the current study, we sought to assess the relationship between psychopathy and local cortical folding in a large sample of incarcerated male offenders using a 3D measurement of folding of the cortical surface, the local gyrification index (LGI; Schaer et al., 2008). We

Table 1
Participant characteristics.

Variable	M	SD
Age	31.80	7.05
IQ	97.44	13.27
Psychopathy checklist-revised		
Total score	22.02	6.95
Factor 1	7.20	3.60
Factor 2	12.74	3.85
Anxiety (STAI-Trait) ^a	40.05	10.69
ASI (total months of use) ^b	228.79	221.54
	%	N
White ^c	47.07	337
Hispanic	28.63	205
Mood Disorder	9.92	71
Anxiety Disorder	6.15	44
Posttraumatic Stress Disorder	1.54	11
Substance Dependence		
Alcohol	31.98	229
Cannabis	32.54	233
Cocaine	29.20	209
Stimulants	24.3	174
Opioids	18.16	130
Sedatives	5.45	39
Hallucinogens	5.45	39
Polysubstance	3.35	24
Antidepressant ^d	10.89	78
Benzodiazepine	0.84	6
Other anti-anxiety	0.27	2
Anti-convulsive/mood stabilizer	2.93	21
Anti-psychotic	0.42	3
Stimulant (prescribed)	0.42	3

^a Participant scores on trait scale of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983). Data missing for 32 participants.

^b No ASI data was available for 125 participants.

^c Data missing for 49 participants, and 120 participants declined to answer.

^d Medication data missing for 43 participants.

predicted psychopathy would be associated with abnormal folding in paralimbic and prefrontal regions implicated in the disorder.

1. Methods and materials

1.1. Participants

Male inmates were recruited from forensic institutions and correctional facilities in Wisconsin and New Mexico where we have ongoing large-scale research projects. Participants had to be between 18 and 45 years old, have an IQ > 70, no history of schizophrenia/schizoaffective disorder, no contraindications for magnetic resonance (MR) scanning, and no history of neurological disorders. A total of 1033 individuals were scanned. For the current study, we only included individuals for whom the following variables were available: structural MRI scan, PCL-R score, estimated IQ, age, and substance dependence assessment (see Table 1 for participant characteristics). Ten subjects were dropped due to failure in preprocessing (i.e., failed segmentation). This yielded a final sample of 716. Written informed consent was obtained from all participants and the study was conducted under the IRB approval from the Ethical and Independent Review Services (E&I) and University of Wisconsin Human Subjects Review Committees.

The Psychopathy Checklist-Revised (PCL-R) was used to assess psychopathy (Hare, 2003). The PCL-R is a semi-structured interview and file review that assesses 20 psychopathy-related items with a rating of (0, 1, 2) and has been shown to be a valid indicator of psychopathy in incarcerated populations (Hare, 2003). In addition to total PCL-R scores, we examined the two primary factor scores. Factor 1 corresponds to interpersonal and affective traits such as callousness and lack of empathy, while Factor 2 scores correspond to antisocial traits and behaviors, such as impulsivity and rule breaking. Twenty-one

participants did not have sufficient data to score Factor 2 independently (due to item-level omissions at the discretion of PCL-R scorers); thus, analyses incorporating Factor 2 are restricted to 695 participants. PCL-R total scores can be prorated to account for omissions (Hare, 2003). Participants' IQ was estimated by the Vocabulary and Matrix Reasoning subtests of the Wechsler Adult Intelligence Scale (Wechsler, 2008). In addition, participants were assessed for the presence of lifetime alcohol or substance use dependence using the Structured Clinical Interview for DSM-IV Disorders (First et al., 2002). For a more granular measurement of substance use, we also conducted our main analysis controlling for number of months of heavy substance use as assessed by a modified version of the Addiction Severity Index (ASI) on a subset of 591 individuals (McLellan et al., 1992).

1.2. Image acquisition

Data were collected on the correctional facility grounds using the Mind Research Network's Siemens 1.5 T Avanto Mobile MRI System with advanced SQ gradients (max slew rate = 200/T/m/s, 345 T/m/s vector summation, rise time 200 ms) and a 12-element head coil. The mobile scanner was transported to each facility and every participant was scanned using the same system. High-resolution ($1 \times 1 \times 1.33$ mm) whole brain multi-echo T1 structural scans (TR = 2530 ms, TE = 1.64, 3.5, 5.36, 7.22 ms, 128 interleaved sagittal slices, no gap, 7° flip angle, and 24-cm FOV) were obtained for each participant.

1.3. Local gyrification index computation

Freesurfer 5.3 (www.nmr.mgh.harvard.edu/freesurfer) was used to reconstruct the cortical surface as a mesh of triangles. Details of the surface reconstruction are described extensively in previous publications (Dale et al., 1999; Fischl et al., 1999). Briefly, the Freesurfer surface reconstruction pipeline consisted of a Talairach transformation to map to Montreal Neurological Institute (MNI) space, normalization of intensity, removal of non-brain tissue (including the skull), and segmentation of gray and white matter.

The local gyrification index (LGI) developed by Schaer and colleagues (Schaer et al., 2008) was used to assess the ratio of buried to visible cortex. This technique is a 3D extension of the gyrification index technique of Zilles et al. (1988), but the new method is fully automated, and therefore, less subjective. Moreover, the LGI technique allows for quantification of gyrification in specific regions (Schaer et al., 2008). The standard LGI computation was followed and involves the creation of a triangulated mesh of the outer hull of the brain, in which a spherical 25 mm region of interest (ROI) is centered at each vertex. In the center of each ROI the LGI is calculated by taking the ratio between the pial surface and outer hull surface. The values are then propagated onto the pial surface, assigning LGI values to each vertex on the mesh surface (Schaer et al., 2008; Schaer et al., 2012).

1.4. Statistical analyses

The relationship between LGI and psychopathy was assessed utilizing a general linear model in a vertex-wise whole-brain analysis. We analyzed the relationship between LGI and PCL-R total scores, as well as the two PCL-R factor scores all in separate regression models using Qdec in Freesurfer. Age, IQ, and diagnosis of alcohol or substance dependence (coded yes or no) were added in as covariates. Substance dependence status was included in the model due to recent research highlighting the potential impact of chronic substance use on cortical morphology (Lisdahl et al., 2013; Mata et al., 2010; Pfefferbaum et al., 2001; Shollenbarger et al., 2015). We also present results without controlling for substance dependence. Each subject's LGI values were first applied to a normalized template (FSaverage). We did not apply any further smoothing given the inherent smoothness of LGI data

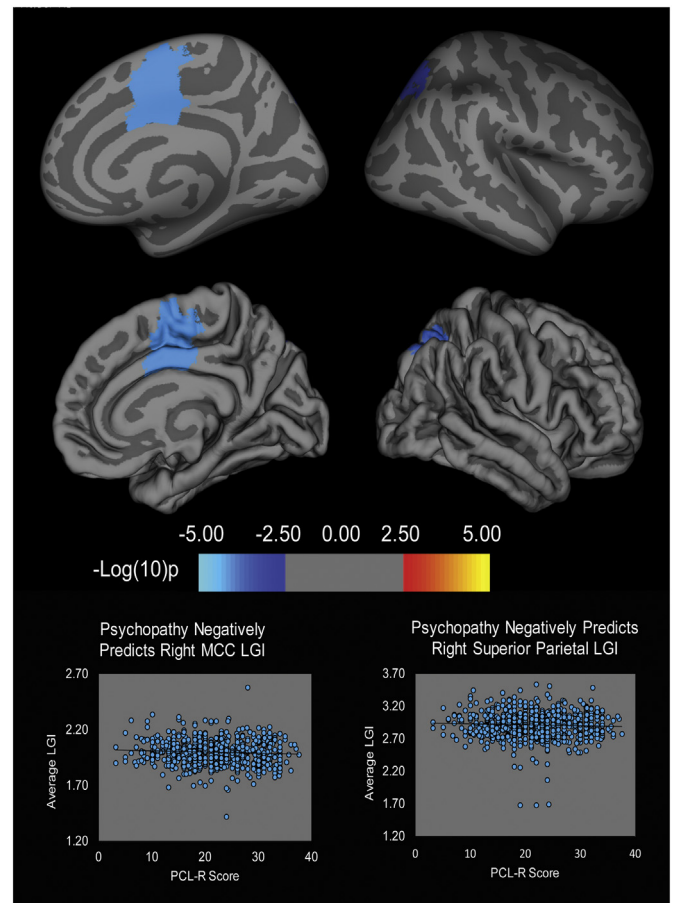


Fig. 1. Negative associations between PCL-R scores and cortical gyrification. Vertex-wise analyses of PCL-R scores demonstrated a negative relationship with cortical gyrification in the midcingulate cortex and superior parietal cortex in the right hemisphere. The top and bottom left brain images show the medial inflated (on the top) and non-inflated (on the bottom) view of the right hemisphere. Below is an image of the plot of average LGI within the MCC cluster and PCL-R scores. The top and bottom images on the right show the lateral inflated (on the top) and non-inflated (on the bottom) view of the right hemisphere, with the plot of the average LGI in the superior parietal cortex cluster and PCL-R scores below.

(Schaer et al., 2013). Cluster correction to control for multiple comparisons was done using Monte Carlo simulation with 10,000 iterations with a vertex-wise threshold of $p < 0.05$. Importantly, Qdec reports transformed Talairach coordinates.

2. Results

The vertex-wise analysis revealed negative associations between PCL-R scores and LGI in several brain areas. Specifically, PCL-R scores negatively predicted LGI values in two clusters in the right hemisphere including the midcingulate cortex (MCC), extending into dorsomedial frontal cortex, (cluster size = 1558.94 mm², points (vertices) = 3635, Talairach coordinates: $x = 3.8$, $y = -2.8$, $z = 31.7$, cluster-wise p value = 0.0001, partial correlation coefficient = -0.11 ; Fig. 1) and lateral superior parietal cortex (cluster size = 1013.07 mm², points = 1937, Talairach coordinates: $x = 27.8$, $y = -56.6$, $z = 43.7$, cluster-wise p value = 0.0009, partial correlation coefficient = -0.09 ; Fig. 1). There was no positive association between PCL-R scores and LGI values anywhere in the brain. There was also no association between PCL-R scores and LGI in any region of the left hemisphere.

Additionally, we ran analyses without substance dependence as a covariate and found that the MCC cluster in the right hemisphere was

still significant, but the cluster was smaller (cluster size = 1213.74 mm², points = 2877, Talairach coordinates: x = 3.8, y = -2.8, z = 31.7, cluster-wise p value = 0.0001, partial correlation coefficient = -0.09; Fig. S1). The superior parietal cluster was no longer significant when not controlling for substance dependence. We also ran the analyses restricted to a subsample of 591 individuals who had continuous substance use scores (number of months of heavy use) assessed using a modified version of the ASI (McLellan et al., 1992), which may provide a more sensitive way to measure the extent of substance use. Again, we found that the MCC finding was still significant in the right hemisphere (cluster size = 1466.23 mm², points = 3427, Talairach coordinates: x = 3.8, y = -2.8, z = 31.7, cluster-wise p value = 0.0001, partial correlation coefficient = -0.10), but the superior parietal was not (Fig. S2). Running the analyses without IQ as a covariate did not alter the findings. There were no LGI differences found between those with and without a substance dependence diagnosis.

We also assessed the relationship between LGI and each of the two factors of psychopathy. Similar to the total PCL-R data, Factor 1 scores were negatively associated with LGI within the right MCC (cluster size = 1025.33 mm², points = 2451, Talairach coordinates: x = 3.8, y = -2.8, z = 31.7, cluster-wise p = 0.0008, partial correlation coefficient = -0.09; Fig. 2). There was also a positive association between Factor 1 scores and LGI in right occipital cortex (cluster size = 2140.28 mm², points = 2669, Talairach coordinates: x = 19.5, y = -86.9, z = 21.2, cluster-wise p = 0.0001, partial correlation coefficient = 0.09; Fig. 2) and left occipital cortex (cluster size = 1295.83 mm², points = 1734, Talairach coordinates: x = -29.3, y = -86.3, z = 11.7 cluster-wise p = 0.0002, partial correlation coefficient = 0.09). Factor 2 scores were not associated with LGI in either hemisphere.

When examining Factor 1 scores, we did not find any notable differences when removing substance dependence as a covariate (Fig. S3). When we removed substance dependence as a covariate in analyses of Factor 2 scores, we found a significant cluster in the right inferior temporal lobe (cluster size = 861.13 mm², points = 1536, Talairach coordinates: x = 39.2, y = -39.8, z = -16.0 cluster-wise p = 0.0047, partial correlation coefficient = -0.09; Fig. 3). Findings were not impacted when removing IQ as a covariate.

3. Discussion

The current study is the first to examine the extent to which differences in cortical gyrification can be associated with psychopathy in adult male offenders. We used a vertex-wise analysis to examine regions of the brain where gyrification was associated with levels of psychopathy when controlling for variance due to age, IQ, and substance dependence. In our large sample of male inmates, we found that psychopathy was negatively associated with gyrification in the right MCC and right lateral superior parietal cortex. Additionally, Factor 1 scores also negatively correlated with gyrification in the right MCC, suggesting that gyrification in this region may be specifically associated with the interpersonal-affective personality characteristics of psychopathy. Overall, these gyrification data are consistent with previous work highlighting psychopathy-related dysfunction in paralimbic regions, as well as aberrations in regions supporting attention and control processes.

Cortical gyrification within the right MCC and adjacent dorsomedial frontal cortex was negatively associated with psychopathy scores. When broken down by Factor scores, Factor 1 scores also showed this association, whereas Factor 2 scores did not. This may indicate that interpersonal-affective personality characteristics are more closely linked to MCC morphology abnormalities than impulsive-antisocial behaviors. Our findings linking structural abnormalities in the cingulate to levels of psychopathy are mostly consistent with previous research. The presence of cingulate structural abnormalities has been further supported

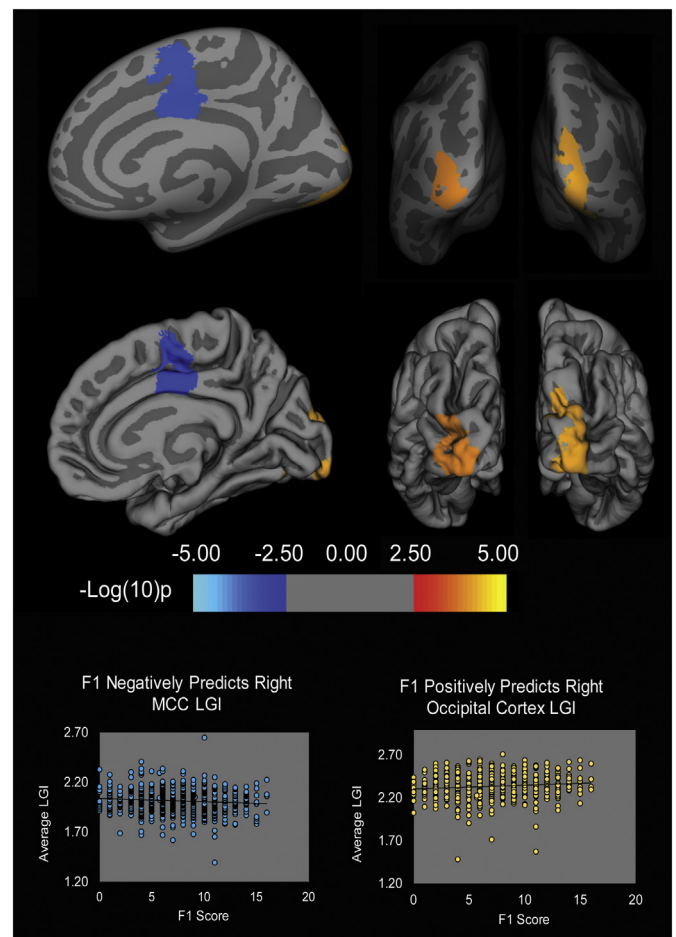


Fig. 2. Positive and negative associations between PCL-R Factor 1 scores and cortical gyrification. Vertex-wise analyses of Factor 1 scores demonstrated a negative correlation with cortical gyrification in the right midcingulate cortex (MCC; blue) and a positive correlation with cortical gyrification in bilateral posterior occipital cortex (orange-yellow). The top and bottom left brain images show the medial inflated (on the top) and non-inflated (on the bottom) view of the right hemisphere. Below these images is the scatterplot of average LGI in the MCC cluster and F1 scores. The top and bottom images on the right show the coronal inflated (on the top) and non-inflated (on the bottom) view of the right and left hemispheres. Below these images is the scatterplot of the average LGI in the right occipital cortex and F1 scores. The relationship between LGI in the left occipital and F1 scores demonstrated a similar relationship.

by recent research demonstrating cortical thinning in the left MCC in psychopathic individuals coupled with decreased functional connectivity between this region and the insula (Ly et al., 2012),¹ a neural circuit that may be important in cognitive control processes (Dosenbach et al., 2007). This implies that structural abnormalities in the cingulate are also linked to cortical connectivity in this population. Furthermore, a recent study published on a subsample of the current sample ($n = 155$) demonstrated psychopathy scores negatively predicted functional connectivity between MCC and lateral parietal cortex (Philippi et al., 2015),¹ which may suggest these aberrations in cortical morphology partly underlie dysfunction within the frontoparietal and cingulo-opercular networks as well. Consistent with the current findings, Hyatt et al. (2012) found youth with conduct disorders demonstrated reduced gyrification in the rostral anterior region of the cingulate compared to controls (see also Fairchild et al., 2015; Smaragdi et al., 2017; Wallace et al., 2014), supporting the presence of folding

¹ Article includes a subsample of the current sample.

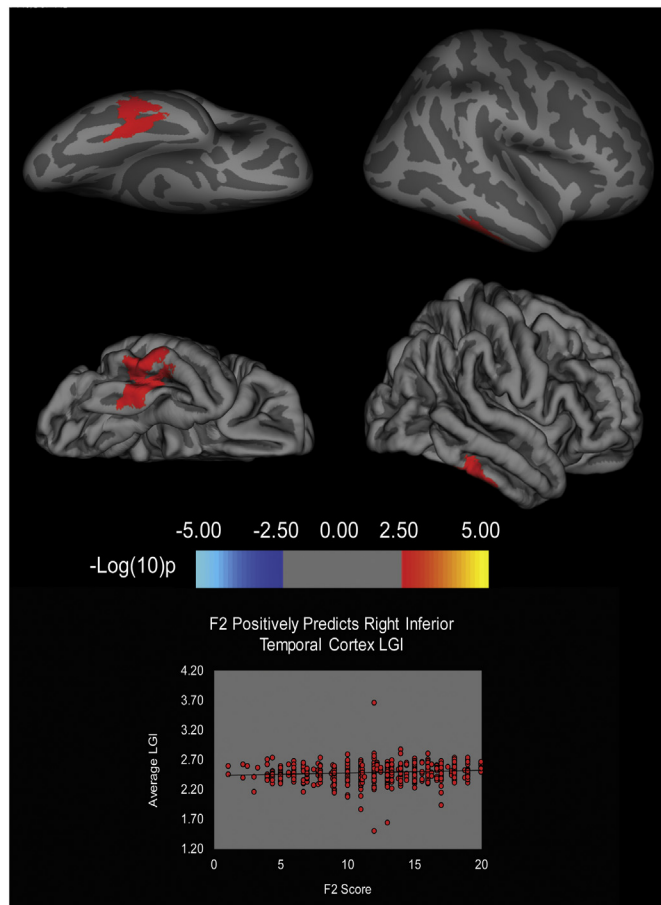


Fig. 3. Positive associations between PCL-R Factor 2 scores and cortical gyrification when not controlling for substance dependence. Vertex-wise analyses of Factor 2 scores demonstrated a positive correlation with cortical gyrification in the right inferior temporal cortex that was not significant when controlling for diagnosis of alcohol/substance dependence. The left images show the bottom inflated (top) and non-inflated (bottom) view of the right hemisphere. The right images show the lateral inflated (top) and non-inflated (bottom) view of the right hemisphere. The bottom image is the scatterplot demonstrating the relationship between average LGI in the right inferior temporal cortex cluster and F2 scores.

abnormalities in the cingulate early in development. Finally, Müller et al. (2008) found reduced gray matter volume in the MCC in psychopathic versus non-psychopathic individuals. However, Glenn et al. (2010) found no relationship between psychopathy in cingulate gray matter volume in a community sample. These discrepancies are likely due to differences in samples, for Müller et al.'s (2008) findings were in a population of forensic inpatients whereas Glenn et al. (2010) used a community sample.

A number of studies have linked dysfunction in psychopathy to the rostral cingulate, which is implicated in affective processing and empathy (Shirtcliff et al., 2009; Vogt, 2005). The MCC plays an important role in cognitive and behavioral control processes that are disrupted in psychopathy, such as error monitoring, response inhibition, task switching and error detection (Brazil et al., 2009; Bush et al., 2000; Carter et al., 1998; Kiehl et al., 2000; Lapierre et al., 1995; Shackman et al., 2011). Previous research has found reduced activation in the MCC in psychopathic individuals during emotional memory tasks (Kiehl et al., 2001) and emotional processing of negative images (Müller et al., 2003). Research has also highlighted the role of the MCC in processing pain and negative emotions (Vogt, 2005) and psychopathic individuals show reduced MCC activation on tasks involving perception of pain in others (Decety et al., 2013), which may underlie a core deficit in

psychopathic individuals ability to empathize when they cause harm (Blair, 2005).

We also found that psychopathy scores were associated with reduced gyrification in the right lateral superior parietal cortex. Although increased gray matter volume and concentration have been reported in boys with callous-unemotional traits in this region within the left hemisphere (De Brito et al., 2009), this is the first study to report structural abnormalities in this region associated with psychopathy. This region is implicated in the frontoparietal network (Corbetta and Shulman, 2002), and is functionally linked to attention processes (Corbetta et al., 1995; Posner et al., 1984), as well as working memory (Koenigs et al., 2009; Wager and Smith, 2003). As noted above, psychopathy scores have been associated with reduced functional connectivity between this region and the MCC (Philippi et al., 2015). Anderson et al. (2017) also reported abnormal functional activation in these networks (Anderson et al., 2017), a dysfunction that may be reflected in the cortical morphology. Given the role of this region in attention, this suggests that disruption in this network may be related to the cognitive abnormalities seen in psychopathy (Larson et al., 2013; Newman et al., 2010).

Finally, analyses of gyrification patterns and PCL-R factor scores demonstrated unique associations between gyrification and Factor 1 scores in bilateral occipital cortex. Ly et al. (2012) reported that psychopathic individuals had reduced cortical thickness in the left occipital cortex compared to controls, and Anderson et al. (2017) found abnormal functional activation in occipital regions was associated with Factor 1 scores. Additionally, Tiihonen et al. (2008) found greater white matter volume in violent offenders with antisocial personality disorder. However, all offenders in the sample had diagnoses of substance dependence whereas controls did not (Tiihonen et al., 2008); therefore, it is still difficult to understand if their white matter findings are independent of substance dependence. Interestingly, one previous study has linked reduced white matter integrity in the inferior frontal-occipital fasciculus to antisocial personality disorder (anterior and posterior) and psychopathy scores (anterior portion) in violent offenders (Sundram et al., 2012). This is consistent with the widely accepted tension-based theory of gyrification, which argues that white matter tension drives cortical folding (Van Essen, 1997) and together with our current findings may suggest these structural abnormalities are related to some of the visual processing deficits in affect recognition evident in antisocial populations (Marsh and Blair, 2008). Despite the altered gyrification associated with Factor 1 scores, we did not find any evidence of abnormal gyrification patterns associated with Factor 2 scores when controlling for substance dependence. When we ran the analysis removing substance dependence as a covariate we found a positive association between Factor 2 scores and gyrification in the right inferior temporal cortex. Structural differences in the temporal lobe in psychopathy have been widely reported (e.g. Müller et al., 2008; Yang et al., 2009) and these findings may suggest shared risk factors with substance use.

As with any single study, it is important to note the potential limitations. First, our sample is limited to incarcerated males. Therefore, it is possible that non-incarcerated samples or women may show different patterns of gyrification in relation to psychopathy. Similarly, our primary aim was to examine the continuous relationship between psychopathy and gyrification measures within a forensic sample, and not to compare all inmates with non-inmate controls. To explore whether inmates differ from non-inmates future studies should consider adding a matched non-forensic control group. Replication of the current findings as well as examining this relationship in other samples, especially youth samples, will be necessary to further characterize how psychopathic traits relate to cortical folding abnormalities. Additionally, although significant, the relationships we detected were small (partial correlation coefficients ≤ 0.10), suggesting psychopathy is only a modest predictor of gyrification. It is of course also possible that other variables, such as socio-economic status or levels of violence/aggression, that may be

important to consider in future work. Finally, gyrification is just one property of the cortical structure, future studies may consider examining other cortical measurements or subcortical regions highlighted in prominent models of psychopathy, such as the amygdala (Blair, 2007; Kiehl, 2006).

In summary, the current study provides the first evidence of abnormal patterns of cortical gyrification in psychopathy. This finding is a novel contribution to the existing literature on brain abnormalities in the disorder and suggests a possible neurodevelopmental aberration that may contribute to MCC instantiated impairments in self-regulation processes in psychopathic individuals. Early theories posited that cortical gyrification reflected structural connectivity and cortical organization that takes place in utero (Armstrong et al., 1995; White et al., 2010). However, recent evidence has suggested that there are reductions in gyrification across the brain during adolescence (Alemán-Gómez et al., 2013; Klein et al., 2014; White et al., 2010), a process thought to be related to cognitive development (Klein et al., 2014). Together, this work suggests that the decreased gyrification associated with psychopathy scores likely reflects abnormalities in either early cortical organization or in processes involved in cortical maturation that occur from childhood to emerging adulthood. This is consistent with developmental models of psychopathy that highlight early disruption of neural systems that support normal emotional processing and self-regulation (Blair et al., 2006), as well as a recent model of psychopathy that focuses on impaired integration of neural systems supporting various cognitive and emotional processes (Hamilton et al., 2015). Future research should seek to clarify links between gyrification and measures of structural and functional connectivity to understand how abnormal cortical development may be associated with abnormalities in cortical connectivity, and ultimately how these neural mechanisms influence behavior.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.nicl.2018.06.007>.

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