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Variable Presence of an Evolutionarily New Brain Structure Is Related to Trait Impulsivity

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ABSTRACT

BACKGROUND: Impulsivity is a multidimensional construct reflecting poor constraint over one's behaviors. Clinical psychology research has identified separable impulsivity dimensions that are each unique transdiagnostic indicators for psychopathology. However, despite this apparent clinical importance, the shared and unique neuroanatomical correlates of these factors remain largely unknown. Concomitantly, neuroimaging research has identified variably present human brain structures implicated in cognition and disorder: the folds (sulci) of the cerebral cortex located in the latest-developing and most evolutionarily expanded hominoid-specific association cortices.

METHODS: We tethered these 2 fields to test whether variability in one such structure in the anterior cingulate cortex (ACC)—the paracingulate sulcus (PCGS)—was related to individual differences in trait impulsivity. A total of 120 adult participants with internalizing or externalizing psychopathology completed a magnetic resonance imaging scan and the Three-Factor Impulsivity Index. Using precision imaging techniques, we manually identified the PCGS, when present, and acquired quantitative folding metrics (PCGS length and ACC local gyrification index).

RESULTS: Neuroanatomical-behavioral analyses revealed that participants with leftward or symmetrical PCGS patterns had greater severity of Lack of Follow Through (LFT)—which captures inattention and lack of perseverance—than those with rightward asymmetry. Neuroanatomical-functional analyses identified that the PCGS colocalized with a focal locus found in a neuroimaging meta-analysis on a feature underlying LFT. Neither quantitative folding metric related to any impulsivity dimension.

CONCLUSIONS: This study advances understanding of the neuroanatomical correlates of impulsivity and establishes the notion that the topographical organization of distinct, hominoid-specific cortical expanses underlies separable impulsivity dimensions with robust, transdiagnostic implications for psychopathology.

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Identifying the neuroanatomical correlates of psychopathology is a major goal of clinical and evolutionary neuroscience. Given the extensive evidence that comorbidity is the norm rather than the exception among individuals with mental illness, research has sought to understand transdiagnostic risk variables shared across disorders (1). Generally speaking, impulsivity is defined by trait-like tendencies toward poor constraint over one's behaviors (2,3). A crucial advance in recent decades is the reproducible finding that rather than being a unidimensional construct, impulsivity is a set of separable but overlapping dimensions (2,4,5) with robust but dissociable patterns of covariance with internalizing and externalizing psychopathology (3,6–8). Although there are multiple approaches to measuring impulsivity, thousands of studies have validated self-report measures that distinguish between impulsive responses to states of high emotion and impulsivity scales without reference to emotion. Impulsivity dimensions characterized by losses of self-control during elevated emotional states, such as the Feelings Trigger Actions (FTA) and Pervasive Influence of Feelings (PIF) dimensions, are often highlighted for their transdiagnostic associations with psychopathology (9–11). However, the Lack of Follow Through (LFT) dimension, which is characterized by distractibility and lack of perseverance to one's goals (5), also explains unique variance in numerous externalizing and inattentive symptoms (6,12,13). Therefore, elucidating the shared and unique neuroanatomical underpinnings of FTA, PIF, and LFT is critical for improving the specificity of clinical interventions for these different forms of impulsivity.

Broadly speaking, neurobiological investigations into impulsivity have consistently pointed to the prefrontal cortex (PFC) across species and developmental stages (14,15). In humans, although patients with PFC lesions may present with increased impulsivity (16–18), the neuroanatomical correlates of impulsivity remain quite heterogeneous within the relatively vast human PFC (19). Review articles have not identified reproducible neuroanatomical correlates within the PFC for studies using traditional gray matter volume and cortical thickness measurements (20,21). However, recent studies have indicated that other structural metrics may be better fit to unlock the neuroanatomical correlates of trait impulsivity (22). Our recent investigations identified that emotion-related

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impulsivity dimensions (FTA and PIF) were related to a regional-level quantitative measure of cortical folding (local gyrification) of the orbitofrontal cortex (OFC) (23). Furthermore, regional PFC surface area yielded stronger effects than cortical thickness did with both emotion-related and non–emotion-related impulsivity dimensions in the ABCD (Adolescent Brain Cognitive Development) Study sample (N = 11,052) (24). These promising regional-level findings have set the stage to investigate whether specific PFC structures are linked to emotion-related and non–emotion-related forms of impulsivity.

Hominoid-specific structures, which include many of the folds (sulci) that comprise the cerebral cortex, are appealing candidates because they may serve as biomarkers for human-specific aspects of cognition—especially in brain regions such as the PFC that have expanded substantially throughout evolution. Crucially, focusing on sulci circumvents contentions regarding analogous and homologous PFC areas across species for two main reasons (22). First, there are contentions regarding what criteria are ideal to define specific brain areas in association cortices. Second, while cerebral cortices vary substantially in size and folding across species, comparing brain areas across species using multiple criteria is useful to determine potential analogous and homologous areas despite these differences. Nevertheless, the parcellation of the cerebral cortex into areas can vary depending on the methods used. For example, Brodmann parcellated the cerebral cortex into 52 areas based on cytoarchitecture (25), while his mentors, the Vogts, parcellated the cortex into 180 areas based on myeloarchitecture (26). The Vogts included sulci, while Brodmann did not. Contrary to this variability and the potential moving boundaries of cortical areas based on the data used, sulci do not move [and a majority of the cerebral cortex is buried in sulci (27–30)]. Thus, sulci may serve as stable landmarks to measure both cross-sectionally and longitudinally across various age groups and clinical populations, and importantly, sulcal morphology and the presence or absence of sulci has been linked to different aspects of cognition in these groups (30–44).

Accordingly, the primary aim of this study was to precisely tease apart specific prefrontal structures that are also identifiable in nonhuman hominoids and that map onto distinct dimensions of impulsivity in humans. One method that is well matched to achieve this goal is the use of high-resolution cortical reconstructions generated from T1-weighted magnetic resonance imaging (MRI) to identify and quantify features of cortical sulci (39,44–46). We recently found that one of the emotion-related impulsivity dimensions (FTA) was related to the depth of specific OFC sulci bilaterally (47), and other groups identified that variations in the sulcal organization of the OFC are altered across multiple disorders (34,48). Here, we extend this previous work by considering variations in the sulcal organization of the anterior cingulate cortex (ACC) and impulsivity. We targeted the ACC given its key roles in emotion-cognition integration (49), which are of relevance for FTA and PIF, and error monitoring and attentional control (50–52), which have been shown to be impaired in the components of impulsivity measured in LFT, lack of perseverance and attention deficits (5,53).

In terms of sulcal organization, the ACC is marked by the consistent cingulate sulcus (CGS) and the variably present paracingulate sulcus (PCGS) (54–56), an evolutionarily new [great ape specific (57,58)] tertiary [emerging in the second/third trimester (59–61)] sulcus above the CGS (Figure 1A). In neurotypical populations, the PCGS is present in at least one hemisphere in approximately 60% to 70% of participants and shows leftward asymmetry, being more common in the left than in the right hemisphere (33,36,54–57). The PCGS also shows more leftward asymmetry in males than females (55,56,62–64). In addition, interhemispheric asymmetry in PCGS presence is related to executive function and can be altered in disorder (31,33,36,65–73). For example, in healthy populations, an asymmetrical PCGS pattern has been associated with better inhibitory control efficiency (31,65–67). In clinical populations, findings can be summarized in the following 4 ways. First, there is increased rightward asymmetry and overall less asymmetry in patients with schizophrenia (68–71). Second, patients with obsessive-compulsive disorder are less likely to have a left hemisphere PCGS (72). Third, patients with bipolar disorder are less likely to have a PCGS in both hemispheres (73). Fourth, PCGS length is reduced and predictive of hallucinations in patients with schizophrenia (32,74). However, it is unknown whether features of the PCGS are trait markers for transdiagnostic features of psychopathology.

To this end, we first documented the intra- and interhemispheric presence of the PCGS in a transdiagnostic adult sample (N = 120, ages 18–55) (Table 1) with varying severity of internalizing and externalizing syndromes that we studied in our prior work (23,47). Then, we explored the relationship between 3 impulsivity factors and variability in PCGS presence. Finally, we assessed whether quantitative features of the PCGS (length) and ACC (local gyrification index [LGI]) were linked to each impulsivity factor.

METHODS AND MATERIALS

Participants

The current study consisted of 120 adults (ages 18–55 years, 66% female) who participated in a parent study that was approved by the University of California Berkeley Committee for the Protection of Human Subjects. Full participant details are documented in Table 1 and in the Supplement.

Behavioral Data Acquisition

Trait impulsivity scores were calculated using the well-validated Three-Factor Impulsivity Index. Factor analyses consistently support 3 impulsivity factors, with strong internal consistency for each scale (5,6,13,75,76). The first factor, FTA, reflects the tendency toward rash action or speech while experiencing overwhelming positively or negatively valenced emotions. FTA consists of 3 subscales: the negative urgency scale (2), positive urgency measure (77), and reflexive reaction to feelings scale (5). The second factor, PIF, is derived from 3 scales that measure cognitive and motivational responses to mostly negative emotion: the generalization (78), sadness paralysis (5), and emotions color worldview (5) scales. LFT measures 2 forms of impulsivity without reference to emotions: the lack of perseverance (2) and distractibility (5) scales. Each factor is measured on a Likert scale from 1 to 5, with higher scores indicating higher severity. Descriptive statistics for the

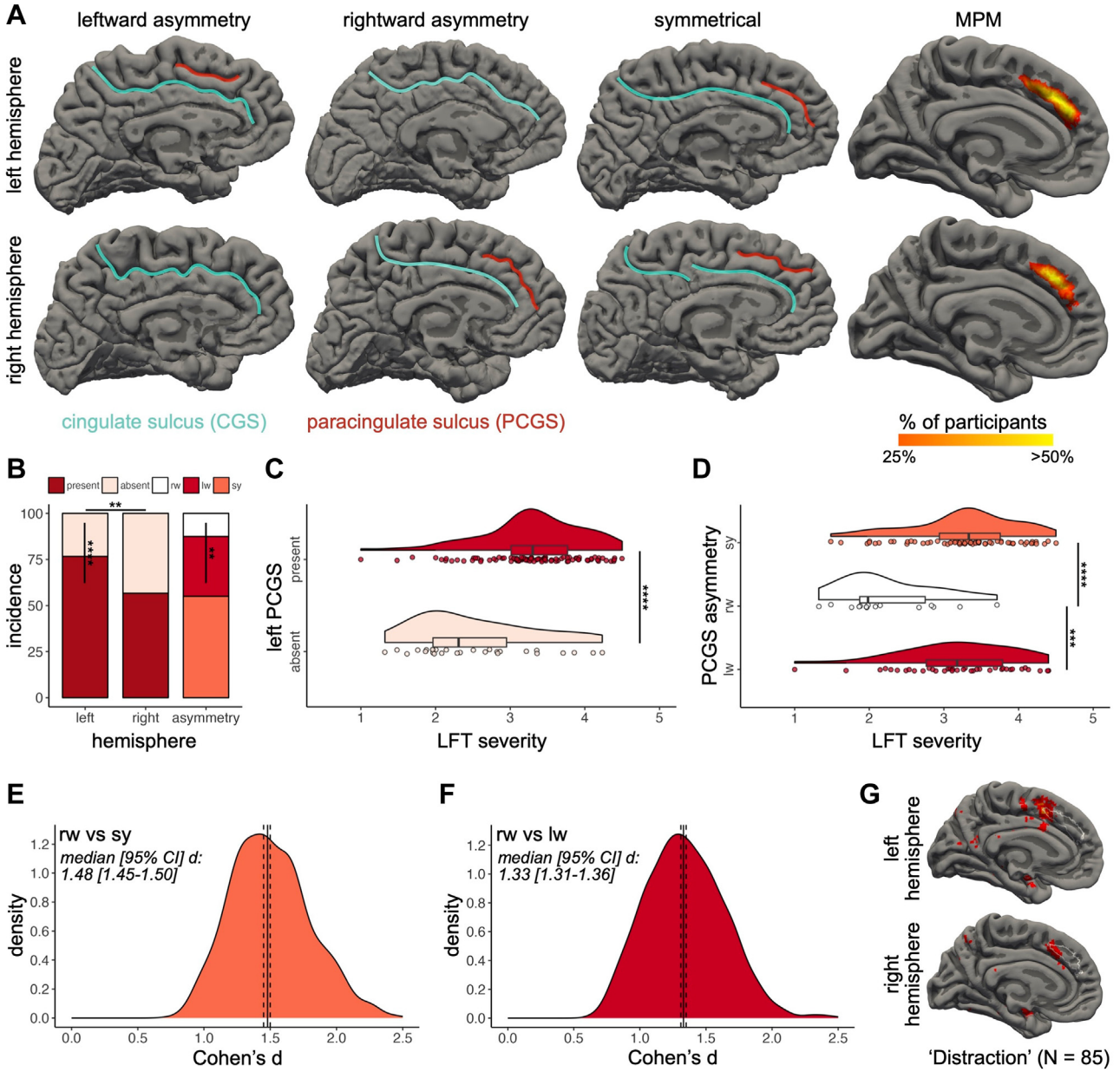

Figure 1. The PCGS is related to nonemotional impulsivity. **(A)** Hemisphere presence/absence (rows) and asymmetry (columns) of the PCGS in 3 example participants in both hemispheres on the wrinkled (pial) cortical surface reconstructions (CGS: blue, PCGS: red). The rightmost column shows the trans-diagnostic maximum probability map (MPM) of the PCGS in both hemispheres (thresholded at 25% overlap for visualization purposes). **(B)** Bar plots showing PCGS incidence (present/absent) and asymmetry rates. **(C)** Raincloud plot (117) showing Lack of Follow Through (LFT) severity as a function of left PCGS presence. **(D)** Same as **(C)** but for PCGS asymmetry. **(E, F)** Distribution of the iteratively sampled effect size (Cohen's *d*) of the significant effects in **(D)** with the median (black line) and 95% CI (dotted lines). **(G)** Pial fsaverage cortical surfaces displaying overlap of a whole-brain false discovery rate–corrected ($p = .01$) uniformity test meta-analysis *z* score map of the distraction term (85 studies; heatmap) and PCGS MPMs (white outline). Asterisks indicate the following *p*-value thresholds: $^{**}p < .01$, $^{***}p < .001$, $^{****}p < .0001$. lw, leftward; rw, rightward; sy, symmetrical.

impulsivity factors are included in Table 1. All impulsivity factors were unrelated to participant age and gender (Table S1).

Imaging Data Acquisition

Participants were scanned using a 3T Siemens TIM Trio MRI scanner (Siemens Healthineers) at the University of California, Berkeley Brain Imaging Center. Sagittal T1-weighted structural images were acquired using a 32-channel receiver head coil and a 6.1-minute magnetization-prepared rapid acquisition gradient-echo sequence. Scanning parameters are as follows: TR = 1900 ms, TE = 2.89 ms, FOV = 256 mm, voxel size = 1-mm^3 isotropic voxels, and parallel acquisition technique

Table 1. Participant Characteristics, *N* = 120

Characteristic	*n* (%) or Mean (SD) [Range]
Gender	
Female	79 (65.8%)
Male	36 (30%)
Nonbinary	5 (4.2%)
Race	
Asian/Asian American	37 (30.8%)
Black/African American	8 (6.7%)
Native Hawaiian/Pacific Islander	1 (0.9%)
Other/multiple races	16 (13.3%)
White/European American	52 (43.3%)
Declined to answer	6 (5%)
Ethnicity	
Hispanic or Latinx	23 (19.2%)
Not Hispanic or Latinx	97 (80.8%)
Age, Years	28.3 (8.6) [18–55]
Education, Years	15.7 (2.3) [12–21]
SCID-5 Lifetime Diagnosis	
Major depressive disorder	97 (80.8%)
Anxiety disorder	81 (67.5%)
Alcohol use disorder	25 (20.8%)
Substance use disorder	24 (20%)
More than 1 disorder	82 (68.3%)
Impulsivity Subtype	
Pervasive Influence of Feelings	3.68 (0.76) [1.92–5]
Feelings Trigger Action	2.81 (0.74) [1.14–4.52]
Lack of Follow Through	3.11 (0.78) [1.00–4.50]

SCID-5, Structured Clinical Interview for DSM-5.

mode = GRAPPA, with acceleration factor PE = 2. Cortical surface reconstructions were generated from the T1-weighted images using FreeSurfer (version 6.0) (79–81). Manual PCGS identification was performed on cortical surface reconstructions in each individual (46,82–84).

Neuroanatomical Data Acquisition

PCGS Identification and Classification Criteria. The PCGS is a variably present sulcus located dorsal and parallel to the CGS (Figure 1A). The PCGS was identified and classified according to its intra- and interhemispheric presence/absence following established criteria (32,38,65,66,68,71,85–88) that are documented in the Supplement. See Figure 1A for examples of intra- and interhemispheric PCGS presence/absence. We also generated probability maps for the PCGS in this transdiagnostic sample (Supplement and Figure 1A).

Relating the PCGS to a Meta-Analysis of Functional MRI Studies Associated with LFT. Although functional MRI research has yet to examine the functional correlates of LFT, to situate the PCGS within putative functional representations of LFT to guide future research, we leveraged the Neurosynth meta-analysis platform (89). We first searched the database for terms associated with LFT [e.g., perseverance (2) and distractibility (5)]. The only term identified was "distraction." Next, we downloaded the uniformity test meta-analysis maps for the search term "distraction" (85 studies). The uniformity test map was generated from a χ^2 test comparing voxel activation in the studies containing the term to the expected activation if it were uniformly distributed across the gray matter. Finally, these maps were projected to fsaverage surface space with the *mri_vol2surf* FreeSurfer function so that the PCGS probability maps could be spatially related.

Quantification and Statistical Analysis

All statistical tests were implemented in R (version 4.1.2) (90). We briefly overview the analyses here; full statistical details are provided in the Supplement.

Qualitative Neuroanatomical Analyses. We began by detailing PCGS incidence against multiple features because this has yet to be documented in a transdiagnostic sample (to our knowledge). We specifically examined whether PCGS incidence differed as a function of 1) hemisphere (within and between), 2) Structured Clinical Interview for DSM-5 lifetime diagnosis, 3) age, and 4) gender. Then, we tested whether each impulsivity index differed as a function of 1) intrahemispheric PCGS presence and 2) interhemispheric asymmetries in PCGS presence.

Quantitative Neuroanatomical Analyses. We implemented 2 parallel analyses to examine the relationship between impulsivity and quantitative features of the PCGS (length, in mm) and ACC subregions (LGI). Given that all results were null, the full methods and statistical analyses, as well as the data and results for these two analyses, are included in the Supplement (including Tables S2–S7 and Figures S1–S3).

RESULTS

A Significant Leftward Bias in PCGS Presence in a Transdiagnostic Sample

Overall, at least 1 PCGS was present in 66% of participants in at least one hemisphere (Figure 1B). The incidence of the PCGS differed significantly between hemispheres (76% of left and 56% of right hemispheres contained a PCGS) ($\chi^2_1 = 9.92$, $p = .001$, 2-sample test for equality of proportions with continuity correction) (Figure 1B). The PCGS was significantly more present than absent in the left hemisphere ($\chi^2_1 = 33.08$, $p = .000000008$, 1-sample proportions test with continuity correction) but not in the right hemisphere ($\chi^2_1 = 1.88$, $p = .17$, 1-sample proportions test with continuity correction). As is common in the field (31,68,71,91,92), we also quantified the interhemispheric asymmetry in PCGS presence (Figure 1A, B). There was a significant leftward bias in PCGS presence in this transdiagnostic sample ($\chi^2_1 = 9.80$, $p = .001$, McNemar's test with continuity correction) (Figure 1B). It has been observed that PCGS asymmetry is reduced in some disorders but not others (36,68–73,93). Therefore, the presence of asymmetry observed here could be due to the wide range of syndrome variability in this transdiagnostic sample (Table 1). However, χ^2 tests (with Yates' continuity correction) identified that intra- and interhemispheric PCGS presence did not vary as a function of any of the Structured Clinical Interview for DSM-5 lifetime diagnoses (Tables S8–S11). Multiple tests were also

performed to evaluate the effects of age and gender on intra- and interhemispheric PCGS presence (Supplement). There were no significant effects (Tables S12 and S13). To aid PCGS identification in nonneurotypical samples, we provide transdiagnostic probabilistic maps with the publication of this article (see Acknowledgments and Disclosures for data and code availability) (Figure 1A).

The PCGS Is Associated With Nonemotional Impulsivity

Then, we sought to relate the presence of the PCGS in each hemisphere separately to each impulsivity index (FTA, PIF, and LFT). An analysis of variance (ANOVA) with factors of left and right PCGS presence revealed a main effect of left PCGS presence for LFT scores ($F_{1,117} = 23.87$, $p = .00001$, $\eta_p^2 = 0.17$) in which the presence of the left PCGS was associated with an increase in LFT severity (Figure 1C). Given the unequal sizes between groups (Figure 1C), we conducted permutation testing, which confirmed the result ($p^*_{n=1000} = .0009$). The fact that there were no other significant effects of PCGS presence on any other impulsivity index tested (Table S14) and the difference in Akaike information criterion (AIC) (Supplement) between the LFT model for PCGS presence and PIF and FTA models ($\Delta AIC_{PIF-LFT} = 13.80$; $\Delta AIC_{FTA-LFT} = 7.45$) indicate that the predictive value of PCGS presence is strongest for LFT.

Next, given the previously documented relationship between interhemispheric PCGS asymmetry and cognitive functioning and disorder (31,33,36,68–71), we ran a second ANOVA to probe the relationship of PCGS asymmetry (symmetrical, leftward asymmetry, and rightward asymmetry) to LFT scores, which revealed a main effect of PCGS asymmetry ($F_{2,117} = 12.69$, $p = .00003$, $\eta_p^2 = 0.18$) (Figure 1D), that was confirmed by permutation testing ($p^*_{n=1000} = .0009$). Post hoc pairwise comparisons showed that participants with a rightward asymmetrical PCGS asymmetry had lower LFT severity than symmetrical ($p = .000002$, $d = 1.47$) and leftward asymmetrical ($p = .0001$, $d = 1.28$) patterns, with no significant differences between symmetrical and leftward asymmetrical patterns ($p = .59$) (Figure 1D). To account for the impact of differences in sample size between the 3 PCGS asymmetry groups on the post hoc effect size tests, we iteratively sampled a size-matched subset of the symmetrical and leftward asymmetrical groups to the rightward sample 1000 times, which confirmed the effect on behavior (Figure 1E, F). No other impulsivity index tested was related to PCGS asymmetry (Table S15) and the difference in AIC (Supplement) between the LFT model for PCGS asymmetry and the PIF and FTA models ($\Delta AIC_{PIF-LFT} = 15.63$; $\Delta AIC_{FTA-LFT} = 9.13$) indicates that the predictive value of PCGS asymmetry is strongest for LFT. Finally, to link the PCGS to LFT functionally, a meta-analysis of neuroimaging research on the term “distraction” (89), a concept related to LFT (5), putatively colocalizes with the PCGS (Figure 1G).

DISCUSSION

Trait impulsivity has shown robust relationships with both internalizing (e.g., depression) and externalizing (e.g., alcohol/substance use) disorders (3,6–8,13). The current findings extend the current picture of the neuroanatomical underpinnings of impulsivity in two key ways. First, they show a regional neuroanatomical dissociation between the nonemotional and emotional impulsivity constructs. Specifically, our previous work (in a largely overlapping sample) identified that OFC folding was related to the severity of emotion-related impulsivity (FTA and PIF) (23,47), whereas the current work identified that non–emotion-related impulsivity (LFT) was related to ACC folding. In combination, these findings highlight that the separability of impulsivity facets in psychological assessment is supported by dissociable neurobiological correlates in the folding of different cortical expanses. This dissociation may be a consequence of these regions being members of different cortical networks—for example, those implicated in “hot” versus “cold” executive function (94). Second, both studies found that rightward asymmetry in folding was associated with reduced impulsivity severity for their respective index, indicating that rightward asymmetry is likely a general neuroanatomical principle that protects against impulsivity—a hypothesis that can be tested in future research. Integrating these points together, future research can seek to examine the sulcal organization of different regions [e.g., the lateral and ventromedial PFC (37,39,44,46,57,95)] to further fill in the neuroanatomical map of impulsivity and determine whether these dissociations (emotional vs. nonemotional impulsivity) and consistencies (rightward asymmetry associated with lower impulsivity severity) hold.

In contrast to our previous work in OFC (23), we identified null relationships between ACC LGI and all impulsivity dimensions. These results indicate that the observed behavioral effects are driven by the neurobiological processes underlying variations of specific sulci and not regional measures of folding (such as LGI). Complementing our recent follow-up study that examined the morphology of specific OFC sulci to impulsivity (47), these results support the notion that these specific impulsivity dimensions are associated with different scales of folding. For example, PIF is related to OFC LGI but not necessarily to the morphology of specific OFC sulci, whereas FTA shows the opposite pattern in the OFC (23,47). Future investigations should seek to identify whether different neurobiological substrates underlie these different measures of cortical folding to provide further insight into the neuroanatomical-behavioral differences observed in this triad of recent studies.

The observation that variability in PCGS presence, but not length, was associated with LFT provides insight into when the neuroanatomical correlates of LFT likely develop. PCGS presence is a cortical feature that forms during the second to third gestational trimesters (59,60,96) and is predominantly determined by in utero environmental factors (33), and the presence/absence of the PCGS does not change after birth (67,97). This is in contrast to quantitative features of sulci (e.g., depth, length, width, and surface area), which change with age (42,63,98,99). These neurodevelopmental differences indicate that, to an extent, features of the fetal environment (in contrast to the postnatal environment) likely play a major role in establishing this neuroanatomical-behavioral effect (36). Longitudinal studies are necessary to further explore the differential impact of these two environments on the currently documented (and to be uncovered) neuroanatomical correlates of LFT.

Translationally, these results extend the growing literature supporting relationships between PCGS presence and multiple psychiatric disorders [e.g., schizophrenia (68–71), obsessive-compulsive disorder (72), and bipolar disorder (73)] by showing that this cortical feature is also associated with LFT, a transdiagnostic predictor of psychopathology, and not only with the disorders themselves. We observed direct effects of LFT, but not of psychiatric diagnoses, on PCGS presence (Tables S8–S11), indicating that these cortical features may be more sensitive indicators of this preclinical trait than diagnoses. This is key given that LFT has been tied to diagnoses and real-world preclinical outcomes, such as engaging in risky behavior (100). One possibility is that PCGS presence provides an early indicator of a continuum of impulsivity risk, which then may be expressed as psychiatric diagnoses in the context of other risk variables. This idea will require further testing given that statistical power in the current study was limited by the imbalanced distributions of those with and without a diagnosis (Table 1).

These findings also hold relevance to understanding the etiology of specific disorders. For example, these findings extend a functional neuroimaging meta-analysis indicating attention-deficit/hyperactivity disorder (ADHD)–related hypoactivation only in the right paracingulate cortex during attention tasks (101). Because the current findings show that rightward paracingulate asymmetry is protective against severe LFT, and previous research indicates that 1) LFT is strongly associated with inattentive symptoms in ADHD (12) and 2) PCGS presence/absence alters ACC functional activity (102–104), future investigations can tether these threads to assess whether the PCGS is functionally and behaviorally implicated in ADHD severity.

In another vein, these results build on studies in neurotypical samples that have demonstrated a link between PCGS presence and cognitive performance by showing that these relationships extend to transdiagnostic samples. These prior studies identified that asymmetry in PCGS presence (either asymmetry in general or in some cases leftward asymmetry) was associated with better performance on multiple cognitive measures [e.g., inhibitory control (31,65–67), fluid intelligence (33), reality monitoring (105), and verbal fluency (63)]. In the current study, we found that rightward asymmetry was associated with lower LFT severity. The anatomical and functional differences in the brains of individuals with different asymmetries in PCGS presence are not well understood (36), which highlights the need for future research to elucidate the mechanisms underlying these observed relationships.

Of course, the reader may also be asking, how could sulcal patterning be mechanistically related to impulsivity? A potential answer lies in the empirical and theorized link between sulci and underlying cortical anatomy and function [reviewed in (22,36,106)]. It has been proposed that PCGS presence could reflect strengthened or otherwise altered local connectivity within the paralimbic cortex (Brodmann area 32) and neighboring regions (Brodmann areas 6, 8, and 9), with implications for vulnerability to various disorders related to ACC function (38,68,71,86). Previous research with neurotypical samples has documented that PCGS presence is related to changes in the local cytoarchitectonic organization of gray matter (54,107), structural and functional connectivity (108,109), and brain function (102–104). In disease, it has been suggested that right PCGS presence may be protective against disease-related neurodegeneration in behavioral-variant frontotemporal dementia by altering local connectivity patterns, thereby leading to a delay in disease onset (38,86). Similarly, studies of PCGS presence in schizophrenia have proposed that lower left PCGS sulcation observed in the disorder may be related to weaker ACC connectivity; thus, increased left PCGS sulcation may be protective (68,71). We propose that a similar mechanism is at play in which local connectivity patterns associated with rightward PCGS asymmetry may be protective—that is, may be related to lower LFT. However, direct investigation of this hypothesis is warranted, and it is important to note that structural and functional network alterations related to PCGS presence are likely only one of many features that play a role in impulsivity.

Although the neuroanatomical precision of the current study is an undeniable strength, it is not without its limitations. Specifically, the time-intensive nature and neuroanatomical expertise needed to manually identify sulci lead to two key limitations. First, the sample size is often limited (46,67,68,83,95,110), necessitating follow-up confirmatory analyses. Although automated methods are being developed to address this limitation, these methods still fall short of manual identification in accuracy (111–114). For example, current automated methodologies identify the PCGS with 70% to 80% accuracy (111,112). Therefore, the current best approach is one integrating multiple methods, such as manual definition guided by probabilistic maps and automated labels. Second, manually defining sulci often limits studies to one region or one sulcus, limiting the observation of more complex interactions between sulci within and between regions on behavior (36). Accordingly, it is necessary to investigate the neuroanatomical correlates of impulsivity in other cortical expanses where the sulcal organization is cognitively and functionally relevant [e.g., the lateral PFC (39,44,67), ventromedial PFC (57,95), and lateral parietal cortex (84,110,115)]. Finally, the functional relationship observed in Figure 1G is limited by 2 features. The first is the inherent limitation of relating 2 group-level probabilistic locations together because this may not fully represent the individual-level relationship (116). The second is that local folding, cytoarchitecture, and functional features of the ACC are all impacted by PCGS variability (54,103,104,107–109). Therefore, we suggest that future research examining the functional correlates of LFT should 1) be done at both the individual and group levels, 2) consider the location of the PCGS as a landmark for functional activity when present, and 3) consider how functional activity may change with PCGS variability.

Conclusions

The current findings highlight that PCGS patterning is a crucial cortical feature that should be considered in future studies to examine how multiscale anatomical and functional features give rise to psychopathology.

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EHW, SAM, and KSW designed the research. EHW, SAM, GMK, and KSW performed the research. MVE and SLJ contributed neuroimaging and behavioral data. EHW, SAM, MVE, SLJ, and KSW wrote the article. All authors gave final approval to the article before submission.

All data and original code used for the current project are publicly available on GitHub (https://github.com/cnl-berkeley/stable_projects). Any additional information that is required to reanalyze the data reported in this article is available from the corresponding author upon request.

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ARTICLE INFORMATION

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