Published in final edited form as:
J Neuropsychiatry Clin Neurosci. 2018 ; 30(3): 236–241. doi:10.1176/appi.neuropsych.17050090.

Neuroanatomical correlates of impulsive action in excoriation (skin-picking) disorder

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Abstract

Excoriation (skin-picking) disorder (SPD) has similarities to obsessive-compulsive disorder (OCD) and is included within the obsessive-compulsive and related disorders (OCRD) category in DSM-5. Separate neuroimaging and neurocognitive studies in SPD implicate difficulty with motor response inhibition and top-down cognitive control in the disorder. No study, however, has examined the neural correlates of SPD in participants with varying degrees of impulsive motor behavior. This study correlated cortical thickness and volumes of selected subcortical structures with stop-signal task performance in participants with SPD ($N = 15$) and healthy controls ($N = 8$). All participants were free from current psychiatric comorbidity, including OCD. In individuals with SPD, longer stop-signal reaction times were correlated with cortical thinning in the right insula and right inferior parietal lobe, and increased cortical thickness in the left lateral occipital lobe ($p < 0.001$ uncorrected), though these findings did not withstand correction for multiple comparisons. There were no significant correlations between cortical thickness in these three structures and stop-signal reaction times in the control group. This study suggests that structural abnormalities in the insular cortex and parietal and occipital regions may play a role in the pathophysiology of SPD. Further neuroimaging research is needed to understand the neurobiology of SPD and its relationship with other OCRDs.

Declaration of interest: Dr. Grant has received research grants from NIMH, National Center for Responsible Gaming, the American Foundation for Suicide Prevention, the Trichotillomania Learning Center, Brainsway, Takeda, and Psyadon Pharmaceuticals. He receives yearly compensation from Springer Publishing for acting as Editor-in-Chief of the Journal of Gambling Studies and has received royalties from Oxford University Press, American Psychiatric Publishing, Inc., Norton Press, and McGraw Hill. Dr. Chamberlain's involvement in this project was funded by a Wellcome Trust Clinical Fellowship (reference 110049/Z/15/Z). Dr. Chamberlain consults for Cambridge Cognition and Shire. Dr. Odlaug has received research funding from the TLC Foundation for BFRBs and receives royalties from Oxford University Press. He has consulted for and is currently employed by H. Lundbeck A/S. H. Lundbeck A/S had no part in any of the studies mentioned in this paper and did not contribute to this paper in any form. Mr. Blum, Mr. Harries, and Ms. Redden report no financial relationships with commercial interests.

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Introduction

Between 1.4 and 5.4% of people meet DSM-5 criteria for excoriation (skin-picking) disorder (SPD), an obsessive-compulsive related disorder (OCRD) characterized by repetitive and compulsive picking of skin leading to tissue damage {1; 2}. Within the OCRDs, SPD is also categorized as a body-focused repetitive behavior disorder alongside trichotillomania, or hair pulling disorder, suggesting possible shared pathophysiology between these disorders {3}. Even so, important differences between SPD and trichotillomania, including unique neurocognitive profiles, indicate that skin picking and hair pulling are not phenotypic manifestations of a single grooming disorder {4}. One way to better understand SPD and its relationship with other conditions is to probe the association between cognitive findings and neural circuitry involved in the disorder.

Separate neurocognitive and imaging studies implicate frontostriatal circuit dysfunction in SPD. From a cognitive perspective, SPD subjects show greater motor impulsivity (as measured by stop-signal tasks) than both individuals with trichotillomania and healthy controls {4; 5}. Neuroimaging studies, likewise, implicate motor control circuits in the pathophysiology of SPD. In a diffusion tensor imaging study, SPD was associated with white matter abnormalities close to the bilateral anterior cingulate cortex {6}, which is activated during unsuccessful response inhibition {7}. Also, a functional neuroimaging (fMRI) study comparing participants with SPD and healthy controls detected significant hypoactivation of the anterior cingulate cortices as well as the dorsal striatum and right medial frontal regions—structures also involved in generation and suppression of movement {8}. Finally, a structural study has reported that SPD volunteers (compared to trichotillomania participants and controls) showed altered brain volume and cortical thickness in frontal areas implicated in impulse control, including the inferior frontal gyrus, orbitofrontal cortex, and nucleus accumbens {9}. The parahippocampal gyrus was additionally implicated in patients with trichotillomania, but not those with SPD {9}. Thus, defects in frontostriatal pathways that exert top-down inhibitory control may contribute to the repetitive picking behavior that characterizes SPD. It is unknown, however, whether variations in these neuroanatomical substrates correlate with deficits in executive function, response inhibition, or both.

Because only a few studies have explored the neurobiology of SPD, this study evaluated the association between neurocognitive function and structural abnormalities in the neural structures that appear to be implicated in SPD. Based on previous research, we hypothesized that impaired inhibitory control (i.e., worse performance on a stop-signal task) in SPD would be associated with cortical thinning in frontal cortical regions and increased volumes in basal ganglia structures, consistent with a neurobiological model implicating lack of top-down control and excessive sub-cortical drive in the manifestation of this habit disorder {10}.

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Methods

Participants

Men and women aged 18–65 with a primary current diagnosis of SPD, based on DSM-5 criteria, were recruited by media advertisements and referrals. A board-certified psychiatrist (J.E.G.) with expertise in SPD and body-focused repetitive behaviors conducted a structured clinical interview with each participant to confirm the diagnosis.

Inclusion criteria were: (1) current DSM-5 diagnosis of SPD; (2) a minimum score of >16 on the Yale–Brown Obsessive Compulsive Scale modified for Neurotic Excoriation (NE-YBOCS); and (3) picking behavior occurred daily for at least 30 min consistently over the past 12 months.

Exclusion criteria in those with SPD included: (1) unstable medical illness; (2) history of seizures; (3) current pregnancy or inadequate contraception in women of child-bearing potential; (4) current suicide risk; (5) lifetime history of bipolar disorder, dementia, or any psychotic disorder; (6) illicit substance use within 2 weeks of study initiation; (7) history of head injury or neurologic disorders; (8) any contraindications to MRI based on safety screening and clinical history; and (9) an inability to understand or undertake the procedures or an inability to provide written informed consent.

Age- and gender-matched healthy controls were recruited by word of mouth and through media advertisements. All control group participants had no current or lifetime psychiatric disorder according to the Structured Clinical Interview for DSM-IV (SCID-I) {11}, the Minnesota Impulsive Disorders Interview (MIDI) {12}, and DSM-5 criteria for SPD, and no use of psychotropic medications.

All participants were recruited and underwent neuroimaging procedures at either the University of Chicago or University of Minnesota under the same principal investigator. Recruitment criteria and procedures were identical between sites. Participants were recruited from November 2010 through May 2012 at the University of Minnesota and from November 2014 through February 2015 at the University of Chicago.

The Institutional Review Boards of the University of Chicago and University of Minnesota approved the study and consent procedures, which followed the Declaration of Helsinki's ethical principles for medical research involving human participants. After a complete description of study procedures and opportunity to ask questions, participants provided written informed consent and received financial compensation.

Procedures

Clinical assessments—All subjects underwent an initial clinical interview with a board-certified psychiatrist, which included:

Yale–Brown Obsessive Compulsive Scale Modified for Neurotic Excoriation (NE-YBOCS). Severity of skin picking disorder symptoms during the preceding seven days was assessed using the 10-item, clinician-administered NE-YBOCS {13; 14} adapted for

DSM-5. The NE-YBOCS is a modification of the Yale–Brown Obsessive Compulsive Scale (Y-BOCS), a reliable and valid scale for obsessive-compulsive disorder (OCD) {15}. Responses to the 10 items were coded on a 4-point scale and summed to produce a composite score ranging from 0 to 40, with higher scores reflecting greater illness severity.

Skin Picking Symptom Assessment Scale (SP-SAS). The SP-SAS is a 12-item, self-report measure evaluating picking urges, thoughts, and behaviors during the previous seven days. Each item is rated 0 to 4 with a maximum total score of 48. Higher scores reflect greater severity of skin picking symptoms. The SP-SAS has demonstrated good preliminary reliability and validity{16}.

Hamilton Depression Rating Scale (HAM-D). The HAM-D is a valid and reliable, 17-item, clinician-administered scale assessing depressive symptoms; higher scores indicate greater depressiveness {17}.

Hamilton Anxiety Rating Scale (HAM-A). The HAM-A is a valid and reliable, 14-item, clinician-administered scale measuring global anxiety {18}. Responses were coded on a 5-point scale of 0 (not present) to 4 (very severe), where higher scores indicate higher levels of anxiety.

Quality of Life Inventory (QoLI). The QoLI is a valid and reliable, 16-item self-administered measure of life satisfaction across several domains, such as health, self-esteem, money, work, love, friendships, and community {19}.

Cognitive assessments—Neurocognitive testing was performed with a touch-screen computer using paradigms adopted from the Cambridge Neuropsychological Test Automated Battery (CANTAB) (CANTABeclipse, version 3; Cambridge Cognition Ltd.), which is well-validated among diverse clinical populations. The cognitive domains of interest were cognitive flexibility (Intra-Extra Dimensional Set Shift task) and response inhibition (Stop Signal Task). We selected these domains because they have been previously implicated in skin picking disorder {4; 5}.

Intra-Extra Dimensional Set Shift (IED) task. The IED was derived from the Wisconsin Card Sorting Test and measures aspects of behavioral flexibility, specifically adaptability to rule changes. The primary outcome measure was total errors made across the whole task, adjusted for any stages not attempted due to failing an earlier stage. Neuroimaging findings indicate that ED shifting depends on bilateral lateral prefrontal cortex activity {20}.

Stop Signal Task (SST). The SST measures control over prepotent (i.e., habitual or dominant) motor behavior. Participants observed a series of left- or right-facing arrows appearing on a computer screen and were instructed to rapidly press a button corresponding to the direction of each arrow. On a subset of trials (“stop” trials), an auditory tone followed presentation of the arrow, indicating that participants should attempt to suppress their motor response for the given trial and wait for the next arrow to be displayed. The primary outcome measure was the stop signal reaction time (SSRT), defined as the average time taken to suppress a prepotent motor response on 50% of trials. Longer SSRTs indicate poorer response inhibition (i.e., greater motor impulsivity). According to neuroimaging data,

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successful response inhibition depends on a right-lateralized neural network including the inferior frontal gyrus and its associated white matter tracts {21}.

Brain imaging—All participants underwent structural brain magnetic resonance imaging (MRI) at the University of Chicago or University of Minnesota using a 3-Tesla (3T) Philips Achieva Quasar Dual 16 Ch system. Three-dimensional (3D), T1-weighted MPRAGE scan was obtained with imaging parameters: slab orientation = sagittal, FOV 256 × 224 × 176, voxel size 1 mm^3, inversion delay time TI = 900 ms, TR/TE = 8.9/3.7 ms, and flip angle = 8°. All controls and 17 SPD participants underwent neuroimaging procedures at the University of Minnesota; three SPD participants did so at the University of Chicago.

Data Analysis

We compared demographic, clinical, and neurocognitive measures between groups using independent sample *t*-tests and likelihood chi-square tests, as appropriate (specific test indicated in the text). Effect sizes were reported for significant group differences (Cohen's *d*). All data were analyzed using SPSS version 22 (IBM). For group comparisons of baseline variables, statistical significance was defined as $p < 0.01$ to account for multiple comparisons, with no Bonferroni correction.

MRI scans were processed on the University of Chicago Midway computer cluster using previously validated methods implemented by FreeSurfer software (version 5.3; http://surfer.nmr.mgh.harvard.edu) {22; 23}. In short, the FreeSurfer workflow consists of an initial cortical surface stream followed by a volume-based (subcortical) stream. In the surface-based stream, FreeSurfer transforms data sets from individual participants to standard Talairach space {24}, normalizes image intensity {25}, extracts the skull and other outer non-brain tissue from the pial surface {26}, and segments the subcortical white matter and deep gray matter volumetric structures {27}. Subsequently, FreeSurfer reconstructs the pial and white matter surfaces to produce representations of cortical thickness, calculated as the distance between each vertex and its corresponding vertex on the opposite surface {28}. The volume-based stream assigns a neuroanatomical label to each subcortical voxel {29; 30}.

We compared cortical thickness between the skin picking disorder and control groups using FreeSurfer's Qdec interface, across the whole cortical surface, at voxel-wise $p < 0.001$ uncorrected. Regions of significant correlation between cortical thickness and cognitive performance were identified across the whole surface, in each group, using the Qdec interface (voxel-wise $p < 0.001$ uncorrected). The statistical threshold of $p < 0.001$ was selected in light of the relatively small sample sizes in the study.

Additionally, volumes of *a priori* selected subcortical structures (putamen, caudate, nucleus accumbens, and hippocampus) were extracted using FreeSurfer's automated parcellation algorithm. These regions of interest were selected because they have been implicated in previous neuroimaging work of SPD or trichotillomania, a closely related disorder. Potential group differences in the volumes of these subcortical structures were explored by exporting subject-level data from Freesurfer into SPSS v22.0 and using independent sample *t*-tests ($p < 0.05$, uncorrected, two tailed). Correlational analysis (Spearman r_s) was used to identify any

relationships between structural abnormalities identified and cognitive performance, again in each group ($p < 0.05$ uncorrected, two tailed).

Results

Participant characteristics

The study sample included participants with SPD ($N = 20$) and healthy controls ($N = 16$), of whom cognitive data were available for $N = 15$ and $N = 8$, respectively. The groups were well-matched in terms of demographic variables (Table 1). Participants with SPD had significantly greater HAM-D ($p < 0.001$) and HAM-A ($p < 0.001$) scores than controls, though these scores were well beneath threshold for clinically significant mood or anxiety disorders (Table 1). The SPD group also reported significantly lower quality of life, as measured by QoLI scores ($p = 0.002$). The mean NE-YBOCS score was 21.2 ($SD = 5.1$), consistent with moderate overall disorder severity. None of the subjects in the SPD sample had a current psychiatric disorder other than SPD, as diagnosed by the SCID-I.

Two SPD participants were currently taking psychotropic medications, both of whom were taking stable doses for over a year. One subject was taking bupropion (450 mg/day), venlafaxine (225 mg/day), and trazodone (100 mg/prn), and the other was taking lamotrigine (300 mg/day), venlafaxine (375 mg/day), and trazodone (100 mg/prn). A third participant was taking a stable dose of *N*-acetylcysteine (1200mg/bid) for more than one year.

In terms of neurocognitive outcomes, SPD participants ($N = 15$) did not differ significantly from controls ($N = 8$) in terms of performance on the IED (assessing cognitive flexibility) or the SST (assessing response impulsivity) (Table 1). The group differences on IED and SSRT remained statistically non-significant even when HAM-D, HAM-A, and QoLI were controlled for using analysis of covariance models (IED: $F(1,23) = 0.395$, $p = 0.538$; SST: $F(1,23) = 0.842$, $p = 0.371$).

Imaging results

There were no significant differences in cortical thickness between SPD and control groups at $p < 0.001$ uncorrected, or in striatal volumes of interest at $p < 0.01$ uncorrected.

In the SPD participants, two significant clusters were identified in which cortical thickness correlated negatively with SSRTs: the first ($F = -3.301$, $p < 0.001$ uncorrected) was maximal at right insula (Talairach coordinates [96.0, 36.9, 6.1], number of voxels 224); and the second ($F = -3.034$, $p < 0.001$ uncorrected) was maximal at right inferior-parietal lobe ([89.0, 40.0, -63.5], number of voxels 174). One significant cluster was identified in which cortical thickness correlated positively with SSRTs in SPD cases ($F = 3.176$, $p < 0.001$ uncorrected), maximal at left lateral-occipital cortex ([-14.2, -96.0, 17.9], number of voxels 135). These findings, however, did not withstand correction for multiple comparisons using a false discovery rate (FDR)-adjusted $p < 0.05$.

No significant cortical clusters were identified that correlated significantly with SSRTs in the control group, and no such significant clusters were found in either group relating to IED performance. There were also no significant correlations between cognitive task scores and

structural volumes in other *a priori* regions of interest, namely selected basal ganglia structures and hippocampus.

Discussion

This is the first study to examine the relationship between neuroimaging abnormalities and cognitive dysfunction in SPD. We identified several significant relationships between response inhibition and cortical thickness in the SPD group, but not in controls. The key findings were that longer SSRTs in the SPD group were associated with reduced thickness in the right insula and right inferior parietal lobe, and greater thickness in the left lateral occipital lobe. Contrary to our hypotheses, SPD participants did not differ significantly from controls in terms of cortical thickness or striatal volumes, nor did we find any significant correlations between basal ganglia volumes and SSRT in patients. These findings may have implications for understanding the neurobiological basis of SPD.

It may be useful to compare our neuroanatomical findings with those previously reported in obsessive-compulsive disorder (OCD), given that SPD is currently conceptualized as an OC-related disorder (OCRD) in DSM-5. In this study, we identified an association between excess cortical thickness in the left lateral occipital lobe and inhibitory control impairment. These findings are consistent with a previous imaging study finding increased cortical thickness in the left lateral occipital region in individuals with OCD {31}. Results from a large multisite study found reduced (not increased) cortical thickness in the same region among participants with more severe OCD symptoms (as measured by the Y-BOCS), but increased thickness in a secondary analysis of patients with OCD not taking psychotropic medications {32}. These mixed findings may reflect clinical heterogeneity within OCRDs and suggest that non-frontostriatal circuitry may also play a role in the pathophysiology of these disorders.

Our study also found a negative correlation between diminished inhibitory control (i.e., longer SSRTs) and cortical thickness in the right insula and right inferior parietal lobe. Both structures are also associated with decreased cortical thickness in OCD, consistent with our results {32}. Although our study design does not permit causal interpretations, it is plausible that abnormalities in these neural regions may contribute to the repetitive behaviors that characterize SPD. The right insula is consistently implicated in inhibitory control and is required to detect and process behaviorally salient stimuli (i.e., “stop” signals) {33}. In the setting of SPD, a decrease in right insula cortical thickness may disrupt the ability to reduce or stop the maladaptive behavior. Likewise, the inferior parietal cortex is responsible for linking tactile perception and fine finger movements {34}. As such, cortical thinning in the inferior parietal cortex may alter the neural pathways that link tactile exploration and appropriate motor responses, resulting in skin picking at bumpy or uneven sites.

This study has several positive features, namely that it is one of only a few neuroimaging studies in SPD and included participants who were free from psychiatric comorbidities, including OCD. Several limitations, however, should be considered. First, MRI results did not survive multiple testing correction using a false discovery rate (FDR)-adjusted p-value of 0.05. These results are not unexpected in light of our relatively small sample and availability

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of cognitive data for only a subset of participants in each group. The lack of significant group differences on imaging and cognitive measures may reflect limited statistical power. Additionally, given preliminary findings from genetic studies supporting a prominent role for individual-level plasticity in the development of the disorder {35}, future imaging studies of SPD should consider multi-site collaborations to increase sample size and generalizability of results. Second, although SPD participants were free from anxiety and depressive disorders, and scored well beneath threshold for clinically significant mood and anxiety symptoms, their scores on the HAM-A and HAM-D were statistically higher than controls. Third, our neuroimaging findings are correlative and not causal. Longitudinal studies are needed to interpret relationships between neuroimaging-cognitive correlations and skin picking behavior. Finally, some SPD participants were medicated, introducing sample heterogeneity that may have influenced the results. Our study, however, was not powered to detect the effect of medication treatment (if any) on brain structure in SPD.

Conclusions

Our findings suggest that greater motor impulsivity in SPD is associated with structural variation in the insular cortex and parietal and occipital regions. These findings are consistent with previous studies of OCD and suggest that cortical thickness in these regions may play a role in the pathophysiology of SPD. A better understanding of the neuropsychiatry of SPD may have utility for treatment interventions.

Acknowledgments

This work was completed in part with resources provided by the University of Chicago Research Computing Center.

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Table 1

Demographic, clinical, and neurocognitive characteristics of skin picking disorder (SPD) participants and healthy controls

Variable	SPD cases (*N* = 20)	Controls (*N* = 16)	Statistic	*df*	*p*	Effect Size
Age, years	29.5 (9.4)	34.0 (15.6)	1.02*t*	21.9[a]	.32	–
Gender, female, *N*[%]	20 [100]	12 [75.0]	3.38c	1	.07	–
Race/ethnicity, white, *N*[%]	19 [95.0]	13 [81.3]	2.71c	2	.26	–
Marital status, single, *N*[%]	14 [73.7]	11 [68.8]	3.38c	3	.34	–
Education, college graduate or more, *N*[%]	10 [55.6]	12 [75.0]	2.26c	3	.52	–
NE-YBOCS total score	21.2 (5.1)	N/A	N/A	N/A	N/A	N/A
SP-SAS total score	30.8 (5.1)	N/A	N/A	N/A	N/A	N/A
HAM-D total score	4.8 (2.8)	0.5 (1.3)	6.03*t*	26.53[a]	**<0.001**	1.97
HAM-A total score	4.8 (3.1)	0.2 (0.6)	6.33*t*	19.67[a]	**<0.001**	2.06
Quality of Life Inventory (QoLI) *t*-score	33.4 (16.1)	50.4 (10.5)	3.44*t*	32	**.002**	1.25
	(*N* = 15)	(*N* = 8)				
IED total errors (adjusted)	12.1 (5.9)	16.3 (19.7)	0.58*t*	7.67[a]	.58	–
SST stop-signal reaction time (SSRT), msec	166.4 (66.4)	167.9 (17.1)	0.06*t*	21	.95	–

Bold indicates significant at $p < .01$.

Data refer to mean (standard deviation) or number of cases [% of group].

NE-YBOCS = Yale–Brown Obsessive Compulsive Scale Modified for Neurotic Excoriation; SP-SAS = Skin Picking Symptom Assessment Scale; HAM-D = Hamilton Depression Rating Scale; HAM-A = Hamilton Anxiety Rating Scale; IED = Intra-Extra Dimensional Set Shift task; SST = Stop Signal Task; SPD = skin picking disorder; c = chi-square; *t* = t-test.

[a]Equal variance not assumed.