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A cognitive comparison of pathological skin picking and trichotillomania

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ABSTRACT

Objective: Pathological Skin Picking (PSP) and Trichotillomania (TTM) share overlapping comorbidity and phenomenology. The extent to which these disorders share a common cognitive phenotype, however, has yet to be examined. This study sought to compare inhibitory control processes in individuals with PSP or TTM.

Methods: Thirty-one subjects with PSP (mean age 31.2 ± 12.5 years; 93.5% female), 39 subjects with TTM (mean age 35.9 ± 10.7 years; 87.2% female), and 33 matched controls (mean age 31.9 ± 9.9 years; 72.7% female) undertook cognitive assessments using the Stop-Signal Task (assessing response impulsivity) and the Intra-dimensional/Extra-dimensional (ID/ED) Set Shift task (assessing cognitive flexibility). Groups were matched for age, gender, race/ethnicity, and education.

Results: PSP was associated with significantly impaired stop-signal reaction times but intact ID/ED cognitive flexibility compared to controls. TTM occupied an intermediate position in terms of stop-signal reaction times between controls and PSP but did not differ significantly from either group on the ID/ED Set Shift Task.

Conclusion: These results replicate the finding of impaired inhibitory control in PSP but suggest TTM may be heterogeneous with respect to such impairment. Future work should explore possible subgroups in TTM and whether cognitive variables are predictive of treatment outcomes.

1. Introduction

Pathological skin picking (PSP) and trichotillomania (TTM) are debilitating conditions with lifetime prevalence estimated to range from 1.4% to 5.4% (Hayes et al., 2009; Keuthen et al., 2010) and from 0.6% to 3.9% (Christenson et al., 1991; Odlaug and Grant, 2010) respectively. TTM is defined as repetitive hair pulling that leads to significant social impairment. TTM is currently classified as an impulse control disorder [APA, 2000 (American Psychiatric Association, 2000)]. PSP is characterized by repetitive and compulsive picking of skin leading to tissue damage. PSP is currently not included in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) or the International Classification of Diseases (ICD-10), and its relationship to TTM remains unsettled.

PSP and TTM share a common phenomenology including age of onset, gender ratio, and psychosocial dysfunction (Cohen et al., 1995; Wilhelm et al., 1999; Stein et al., 2006; Odlaug and Grant, 2008). Both disorders have also demonstrated an inability to suppress inappropriate repetitive behaviors (Chamberlain et al., 2006; Odlaug et al., 2010). Differences, however, have also been found, such as sensory triggers and time spent engaged in the behavior (Odlaug and Grant, 2008).

Although research has indicated some similarities in cognitive dysfunction shared by PSP and TTM (Chamberlain et al., 2006; Odlaug et al., 2009), these studies were performed in relatively small samples by different investigators and under different testing conditions. To our knowledge, there have been no published comparisons of the two disorders by the same investigators under the same testing conditions. Consequently, this study sought to examine both PSP and TTM to determine cognitive dysfunction. Based on these prior studies, we hypothesized that both disorders would demonstrate motor impulsivity as indexed by the stop-signal paradigm, but intact set-shifting.

2. Methods

The sample consisted of 31 subjects with PSP (mean age 31.2 ± 12.5 years; 93.5% female), 39 subjects with TTM (mean age 35.9 ± 10.7 years; 87.2% female), and 33 healthy controls with no

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psychiatric history (mean age 31.9 ± 9.9 years; 72.7% female). Patients were required to have a current DSM-IV primary diagnosis of TTM or to meet the full proposed diagnostic criteria for primary PSP (Arnold et al., 2001). Healthy controls were required to have no lifetime or current psychiatric illness.

PSP was diagnosed using the following criteria: 1) Damage to the skin as a result of recurrent picking or manipulation of the skin; 2) an increase in tension or unpleasant emotional or physical state immediately preceding the picking episode or when trying to resist urges to pick; 3) feelings of pleasure, gratification or relief at the time of picking; 4) picking results in clinically significant distress or impairment; 5) picking is not a result of a medical condition, substance use, or attributed to another mental disorder (Arnold et al., 2001). In order to obtain a robust sample of PSP subjects, only those subjects who endorsed noticeable skin/tissue damage resulting from picking behavior were included.

Past 12-months co-occurring psychiatric disorders were assessed by board-certified psychiatrists using the Structured Clinical Interview for DSM-IV disorders (SCID) (First et al., 1995).

Subjects were excluded if they were unable to understand and consent to the study procedures or had a history of neurological disorders. The Institutional Review Board of the University of Minnesota approved the studies and the informed consent statements. After study procedures were explained and subjects had the opportunity to ask questions, all study participants provided voluntary written informed consent. All study procedures were carried out under the guidance of the latest version of the Declaration of Helsinki.

2.1. Clinical assessments

PSP and TTM subjects were assessed using the following scales:

Clinical Global Impression-Severity (Guy, 1976). The CGI consists of a reliable and valid 7-item scale used to assess clinical severity in overall symptoms. Using a Likert scale, the CGI ranges in score from 1 = "not ill at all" to 7 = "extremely ill."

Sheehan Disability Scale (*SDS*) (Sheehan, 1983). The SDS is a 3-item, reliable and valid self-report scale that assesses functioning in three areas of life: work/school, social or leisure activities, and home and family life. Scores on the SDS range from 0 to 30 with each question comprising a 0 (no disruption) to 10 (extreme disruption).

Hamilton Anxiety Rating Scale (*HARS*) (Hamilton, 1959). The HARS is a reliable and valid, clinician-administered, 14-item scale that provides an overall measure of global anxiety.

Hamilton Depression Rating Scale (*HDRS*) (Hamilton, 1960). The HDRS is a valid and reliable, 17-item, clinician-administered rating scale assessing severity of depressive symptoms.

Quality of Life Inventory (*QoLI*) (Frisch et al., 1992). The QoLI is a valid and reliable, 16-item, self-administered rating scale, that assesses life domains such as health, work, recreation, friendships, love relationships, home, self-esteem and standard of living. The scale rates each domain by assessing its perceived importance to their overall happiness and how satisfied they are with that particular domain. The QoLI has four ranges from which overall quality of life classification is determined (high, average, low, very low) based upon the calculated T-Score (high: 58–77; average: 43–57; low: 37–42; very low: 0–36) (Frisch et al., 1992). All subjects, including healthy controls, completed the QoLI.

2.2. Cognitive assessments

Subjects undertook paradigms from the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Cambridge Cognition Limited, 2006) quantifying aspects of impulsivity (relating to response inhibition) and cognitive flexibility. Interpretation of the cognitive tasks was performed by a rater blind to group assignment.

2.2.1. Stop-signal test (SST)

The Stop-signal test is a well-validated task quantifying the ability to suppress impulsive responses (Logan et al., 1984; Aron et al., 2004). Subjects observe a series of directional arrows appearing one at a time on a computer screen, and make speeded motor responses depending on the direction of each arrow, with a button box (left or right). On a subset of trials, an auditory beep occurs (the 'stop-signal') which indicates that the subject should try to inhibit their response for that particular trial. The task adjusts the gap between the 'go' and 'stop' signals dynamically depending on the individual's performance, such that the likelihood of successful inhibition over the whole of the task approximates 50%. The primary outcome measure is a sensitive estimate of the time taken by the subject's brain to stop a pre-potent response, referred to as the 'Stop-signal reaction time' (SSRT). Median reaction time for go trials is also recorded, along with the number of directional errors made, and the overall percentage of successful inhibition.

2.2.2. Intra-dimensional/extra-dimensional set shift task (IDED)

The IDED task includes aspects of rule learning and behavioral flexibility, and was derived from the Wisconsin Card Sort Test (Lezak et al., 2004). Through trial and error, and feedback, volunteers attempt to learn a rule about which one of the two stimuli is correct. After each choice, feedback is given ('correct' or 'incorrect'). Once learning criterion is obtained (six consecutive correct responses), the computer changes the rule, and the volunteer must then adapt their behavior appropriately. There are nine stages to the task, requiring different components of set acquisition, reversal, and flexibility. Key measures are the total number of errors made on stages 6 and 8 of the task, corresponding to ID and ED set-shifting respectively.

2.3. Data analysis

PSP subjects, TTM subjects, and healthy controls were compared on demographic and clinical variables and rates of current co-occurring disorders. Between-group differences on demographic, clinical, and cognitive variables were tested using one-way ANOVAs, or chi-square tests, with follow-up post-hoc tests as appropriate (protected Least Significant Difference tests, or further chi-square as appropriate). Where significant group differences were found on a given measure, possible relationships with disease severity (CGI severity, HAM-A, HAM-D scores) were evaluated using Spearman's r. All comparison tests were two-tailed and an alpha level of 0.05 was used to determine statistical significance. For significant pair-wise differences, effect sizes were calculated (Cohen's d) with small (≥0.2–0.3), medium (≥0.5), and large (≥0.8) effect sizes established.

3. Results

Demographic and clinical information on the sample can be found in Table 1. PSP, TTM, and healthy control subjects did not differ significantly on any demographic variable. In terms of clinical variables, TTM subjects had significantly greater CGI Severity scores (4.56 ± 0.75 compared to 4.16 ± 0.52 in PSP; $p = 0.014$) than PSP subjects (corresponding to moderate to marked overall clinical severity). Depressive and anxiety symptoms were low and overall quality of life was "average" for both groups. Both PSP and TTM subjects generally reported moderate psychosocial dysfunction

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Table 1
Demographic and clinical comparison of pathological skin picking (PSP) and trichotillomania (TTM) subjects.

	PSP ($n = 31$)	TTM ($n = 39$)	Healthy Controls ($n = 33$)	Statistic	p-value
Age					
Mean (±SD), years	31.23 ± 12.52	35.85 ± 10.66	31.91 ± 9.88	1.849a	0.163
Gender, n (%)					
Female	29 (93.5)	34 (87.2)	24 (72.7)	5.633c	0.059
Race, n (%)					
Caucasian	29 (93.5)	39 (100)	30 (90.1)	3.443c	0.447
Other	2 (6.5)	0 (0)	3 (9.9)		
Marital, n (%)					
Single/Living Together/Gay	16 (51.6)	18 (46.2)	22 (66.7)	4.995c	0.288
Married	14 (45.2)	18 (46.2)	8 (24.2)		
Divorced/Separated/Widowed	1 (3.2)	3 (7.6)	3 (9.1)		
Education, n (%)					
High School or less	2 (6.5)	4 (10.3)	0 (0)	4.647c	0.233
Some college or more	29 (93.5)	35 (89.7)	33 (100)		
Age of Disease Onset, years	13.48 ± 9.63	13.13 ± 8.4	–	0.165t	0.869
Sheehan Disability Scale	11.94 ± 2.84	11.95 ± 6.07	–	0.011t	0.991
Clinical Global Impressions scale	4.16 ± 0.52	4.56 ± 0.75	–	2.530t	0.014
Hamilton Depression Rating Scale	4.1 ± 2.97	4.67 ± 4.03	–	0.658t	0.513
Hamilton Anxiety Rating Scale	3.94 ± 2.58	4.41 ± 3.28	–	0.659t	0.512
Quality of Life Inventory (QOLI)					
T-Score	42.9 ± 12.56	44.69 ± 11.07	–	0.633t	0.529
Any current comorbid disorder, n (%)	22 (71)	21 (53.8)	–	2.137c	0.144
Currently taking a psychotropic medication, n (%)[a]	15 (48.4)	13 (33.3)	–	1.631c	0.202

Statistic: a = ANOVA; c = chi-square; t = t-test.
All values are Mean (±SD) unless otherwise indicated.
[a] Number of patients receiving specific classes of treatment: PSP: Stimulants ($n = 3$), SSRIs ($n = 8$), mixed reuptake inhibitors ($n = 3$), antipsychotic ($n = 2$), tricyclic ($n = 1$), 5HT 1A agonist ($n = 1$), benzodiazepines ($n = 2$); TTM: SSRIs ($n = 8$), mixed reuptake inhibitors ($n = 7$), tricyclic ($n = 1$). Note many patients were on polypharmacy.

using the SDS. Patients did not differ significantly in the proportion receiving psychotropic medications, and were on a variety of classes of agent (footer, Table 1).

On the IDED task, there were no significant effects of group on the number of errors made on the ID shift ($F = 0.011$, $p = 0.990$) or ED shift stages ($F = 2.249$, $p = 0.111$) (mean ± SD, ID: PSP 0.29 ± 0.59, TTM 0.31 ± 0.47, CON 0.30 ± 0.37; ED: PSP 7.6 ± 8.7, TTM 11.2 ± 10.5, CON 8.7 ± 9.54).

There was a significant effect of group on SSRTs ($F = 3.242$, $p = 0.043$). Post-hoc tests indicated that this group difference was attributable to significantly longer SSRTs in the PSP group (195.82 ± 85.14 ms) as compared to controls (157.49 ± 47.57 ms) ($p = 0.014$; $D = 0.56$). The TTM group (171.00 ± 46.43 ms) did not differ significantly from controls ($p = 0.351$) nor from the PSP group ($p = 0.094$) on SSRT. Correlation analysis indicated a trend toward longer SSRTs being associated with greater disease severity in PSP (CGI severity $r = 0.316$, $p = 0.083$); there were also significant correlations between SSRTs and depressive/anxiety scores in patients with PSP (HAM-D $r = 0.447$, $p = 0.012$; HAM-A $r = 0.457$, $p = 0.010$). No such significant correlations were found in the TTM group (all $p > 0.10$). In a supplementary analysis in patients only, covarying for all disease severity scores, PSP still did not differ significantly from TTM recruits in terms of SSRTs ($p = 0.051$).

There was a significant effect of group on the number of directional errors made ($F = 3.405$, $p = 0.037$). This was due to significantly more directional errors being made in the PSP compared to TTM group (PSP: 4.71 ± 8.88; TTM: 2.08 ± 2.98; $p = 0.0443$) and compared to the controls (PSP: 4.71 ± 8.88; CON: 1.33 ± 1.81; $p = 0.0158$). TTM did not differ significantly from controls on this measure (TTM: 2.08 ± 2.98; CON: 1.33 ± 1.81; $p = 0.570$). There were no significant correlations between directional errors and disease severity measures (CGI severity, HAM-D, HAM-A) in PSP or in TTM subjects (all $p > 0.10$). There was no significant effect of group on the median go response times on the SST task ($F = 0.270$, $p = 0.764$) (mean ± SD: PSP 489.06 ± 157.9 ms, TTM 469.96 ± 141.21 ms, CON 492.78 ± 127.12 ms). There was no significant effect of group on the overall probability of successful inhibition [p(inhibit)] ($F = 1.885$, $p = 0.157$) (mean ± SD: PSP 0.52 ± 0.10, TTM 0.56 ± 0.08, CON 0.53 ± 0.08) Table 2.

Table 2
Performance on cognitive tasks in pathological skin picking and trichotillomania versus healthy controls.

	Performance (mean ± SD)			ANOVA		Post-Hoc LSD Tests		
	PSP ($n = 31$)	TTM ($n = 39$)	Healthy Controls ($n = 33$)	F	p	PSP vs. Controls P	TTM vs. Controls P	PSP vs. TTM P
IDED ID errors	0.29 ± 0.59	0.31 ± 0.47	0.30 ± 0.37	0.011	0.990	–	–	–
IDED ED errors	7.6 ± 8.7	11.2 ± 10.5	8.7 ± 9.54	2.249	0.111	–	–	–
SST median go reaction time	489.1 ± 157.9	469.9 ± 141.2	492.8 ± 127.1	0.270	0.764	–	–	–
SST SSRT	195.8 ± 85.1	171.0 ± 46.4	157.5 ± 47.6	3.242	0.043	0.014	0.351	0.094
SST directional errors	4.71 ± 8.88	2.08 ± 2.98	1.33 ± 1.81	3.405	0.037	0.016	0.570	0.044
SST p(inhibit)	0.52 ± 0.10	0.56 ± 0.08	0.53 ± 0.08	1.885	0.157	–	–	–

T = t-test: two sample assuming equal variances; P = two-tailed t-test.
Abbreviations: LSD = Least Significant Difference; IDED = Intra-dimensional Extra-dimensional; SST = Stop-Signal Task; SSRT = Stop-Signal Reaction Time.

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4. Discussion

This is the first study to directly compare cognitive function in PSP and TTM, two debilitating and yet under-researched psychiatric conditions. Both PSP and TTM have been proposed as part of a spectrum of compulsive disorders associated with inhibitory control deficits manifesting as excessive motoric output (Chamberlain et al., 2005). Previous studies have shown that PSP and TTM may share overlapping deficits in motor inhibition or the tendency to suppress pre-potent motor responses (Odlaug et al., 2010; Chamberlain et al., 2006), a deficit that implicates distributed circuitry including the right inferior frontal gyrus and bilateral anterior cingulate cortices (Aron et al., 2004; Hampshire et al., 2010). In the current study, we identified significant inhibitory deficits only in the PSP subjects, while TTM subjects occupied an intermediate position between controls and PSP on SSRTs (not differing significantly from either controls or the other clinical group in performance). The groups did not differ significantly on set-shifting, which is thought be dependent on the dorsolateral prefrontal cortices (Owen et al., 1991; Hampshire and Owen, 2006).

When compared to the previous literature (Chamberlain et al., 2006), these findings suggest several possible interpretations. First, although PSP and TTM share an overlapping phenomenology (Odlaug and Grant, 2008; Odlaug et al., 2010; Stein et al., 2010), the two behaviors may actually differ in terms of pathophysiology, or the inhibitory deficit may be more extreme in PSP. Second, some individuals with PSP may share a common pathophysiology with some individuals with TTM, but the behaviors may reflect greater etiological heterogeneity than previously thought. Finally, the cognitive tasks that have been used to examine these behaviors may not reflect the underlying deficits giving rise to these behaviors. A greater number of tasks with broader examination of cognitive domains (including other aspects of impulsivity, such as measured by delay-discounting tasks) may have detected more commonalities that in turn explain the large phenomenological overlap in these disorders.

There are several limitations that must be noted in this study. First, the non-ethnically diverse sample represented may misrepresent PSP and TTM subjects in the overall general population. Future studies should obtain a more ethnically diverse study population to validate these results. Second, there is possibility that the cognitive tasks reflect pathology other than the compulsive behaviors. Although we did not administer the HARS or HDRS to our sample of healthy controls, all subjects underwent an extensive clinical interview and scores for both the HARS and HDRS were relatively low in the PSP and TTM populations. It is unlikely therefore that the differences in cognitive assessment reflect underlying anxiety or depression. A majority of subjects had a current co-occurring psychiatric disorder that may have affected the cognitive testing, and many were receiving psychotropic medication(s). However, in order to be involved in the research study, PSP or TTM had to be the primary psychiatric condition. In a supplementary analysis, we found that the SSRT performance of patients within each clinical group on psychotropic medication(s) were similar to those within each group who were not (numerically and statistically). It should be noted that this study was neither designed nor powered to address this issue, however. Third, the samples were relatively small and to some extent numerically unbalanced. Larger, more balanced samples may be needed to detect significant differences between them. Fourth, this study was not powered to assess subtypes of PSP and TTM that may share greater overlap in cognitive deficits. Finally, although the Stop-Signal and ID/ED tasks are reliable and valid measures of inhibitory control and cognitive flexibility, respectively, the subjects were not actively engaged in picking or pulling at the time of testing. These tasks therefore may not fully capture the cognitive deficits during acute episodes of picking or pulling.

Despite these limitations, this study does have several strengths. First, although the sample size may be overall low, for disorders such as PSP and TTM which lack a significant amount of information, our sample size was quite large. Second, all subjects in this study were interviewed directly by a board-certified psychiatrist with extensive knowledge of both PSP and TTM. Finally, the cognitive tasks used in this study have been validated in previous research. Future research should examine whether certain subtypes of both PSP and TTM (for example, automatic versus focused, early onset versus onset, etc) exhibit more consistent cognitive findings, and whether any deficits identified are responsive to successful pharmacological intervention with pre-existing and novel agents.

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Contributors

Dr. Grant and Mr. Odlaug designed the assessment and interviewed the subjects described in the sample. Mr. Odlaug managed the literature searches and compiled the database. Dr. Chamberlain performed the statistical analyses. Dr. Grant and Mr. Odlaug wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Conflict of interest

Dr. Grant has received research grants from NIMH, NIDA, National Center for Responsible Gaming and its affiliated Institute for Research on Gambling Disorders, and Psyadon Pharmaceuticals. Dr. Grant receives yearly compensation from Springer Publishing for acting as Editor-in-Chief of the Journal of Gambling Studies. Dr. Grant has received royalties from Oxford University Press, American Psychiatric Publishing, Inc., Norton Press, and McGraw Hill. Dr. Chamberlain has consulted for Cambridge Cognition, P1Vital, and Shire Pharmaceuticals. Mr. Odlaug has received honoraria from Oxford University Press and Current Medicine Group, LLC.

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