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A neuropsychological comparison of obsessive–compulsive disorder and trichotillomania

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Abstract

Background: Obsessive–compulsive disorder (OCD) and trichotillomania (compulsive hair-pulling) share overlapping co-morbidity, familial transmission, and phenomenology. However, the extent to which these disorders share a common cognitive phenotype has yet to be elucidated using patients without confounding co-morbidities. *Aim*: To compare neurocognitive functioning in co-morbidity-free patients with OCD and trichotillomania, focusing on domains of learning and memory, executive function, affective processing, reflection-impulsivity and decision-making. *Method*: Twenty patients with OCD, 20 patients with trichotillomania, and 20 matched controls undertook neuropsychological assessment after meeting stringent inclusion criteria. *Results*: Groups were matched for age, education, verbal IQ, and gender. The OCD and trichotillomania groups were impaired on spatial working memory. Only OCD patients showed additional impairments on executive planning and visual pattern recognition memory, and missed more responses to sad target words than other groups on an affective go/no-go task. Furthermore, OCD patients failed to modulate their behaviour between conditions on the reflection-impulsivity test, suggestive of cognitive inflexibility. Both clinical groups showed intact decision-making and probabilistic reversal learning. *Conclusions*: OCD and trichotillomania shared overlapping spatial working memory problems, but neuropsychological dysfunction in OCD spanned additional domains that were intact in trichotillomania. Findings are discussed in relation to likely fronto-striatal neural substrates and future research directions.

Keywords: Obsessive; Compulsive; Impulsive; Strategy; Fronto-striatal; Spectrum; Cognition

1. Introduction

Obsessive–compulsive disorder (OCD) and trichotillomania (repetitive hair-pulling) are debilitating conditions with lifetime prevalence estimated to be 2–3% (Robins et al., 1984) and 0.6% (Christenson, Pyle, & Mitchell, 1991), respectively. OCD is currently classified as an anxiety disorder (DSM-IV, 1994) and is characterised by recurrent intrusive thoughts (obsessions) and/or repetitive mental or behavioural rituals performed in response to obsessions or according to rigid rules (compulsions). Trichotillomania is characterised by repetitive hair-pulling that leads to significant social impairment, and is currently classified as an impulse control disorder (DSM-IV, 1994). However, this classification may be problematic, given the association between trichotillomania and compulsive self-injurious symptoms such as skin picking (Lochner et al., 2005). OCD and trichotillomania share overlapping co-morbidity, familial transmission, and possibly treatment response (Hollander & Rosen, 2000; Stein, Simeon, Cohen, & Hollander, 1995). Phenomenologically, both are characterised by difficulties suppressing inappropriate repetitive behaviours, suggesting underlying dysregulation in inhibitory control processes (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). However, whereas hair-pulling is a relatively simplistic behaviour, rituals in OCD are often complex and performed in response to obsessional thoughts or according to rigid rules (DSM-IV, 1994). Thus, while

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both OCD and trichotillomania may share overlapping difficulty inhibiting motor behaviour, problems with higher level rigidity may be restricted to OCD alone (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2006b).

Many studies have examined neuropsychological functioning in OCD, but relatively little research has been conducted in trichotillomania. Deficits in OCD have been reported on tests of executive planning, strategy implementation, and memory (reviewed in, e.g. Chamberlain et al., 2005; Fontenelle, Mendlowicz, Paulo, & Marcio, 2006; Kuelz, Hohagen, & Voderholzer, 2004). For trichotillomania, case–control studies find some support for deficits on tests of memory, executive function, and divided attention (Coetzer & Stein, 1999; Keuthen et al., 1996; Stanley, Hannay, & Breckenridge, 1997). Very few studies have attempted to compare cognition in OCD and trichotillomania directly. Of the handful of available studies, two found tentative support for an overlapping visuo-spatial memory deficit (Coetzer & Stein, 1999; Rettew, Cheslow, & Rapoport, 1991), and another found evidence for differential impairments on the object alternation test (impaired in trichotillomania) and the Wisconsin card sorting test (impaired in OCD) (Bohne et al., 2005). Existing studies have frequently included patients with co-morbidities (especially, depression and other anxiety disorders), which likely contributed to the cognitive findings, and have assessed a relatively narrow range of neuropsychological function.

In recent years, theoretically driven computerized tests have been developed that are capable of tapping separable cognitive domains dependent upon fronto-striatal circuitry (Chamberlain & Sahakian, 2005). Modern models of OCD neuropathology posit abnormalities in fronto-striatal circuitry (especially anterior cingulate cortex, orbitofrontal cortex, and basal ganglia) (Chamberlain et al., 2005; Graybiel & Rauch, 2000). Whether or not similar neural circuitry is implicated in trichotillomania is unclear. The present study sought to compare performance of axis-I co-morbidity-free OCD and trichotillomania patients using a battery of tests targeted on likely areas of impairment. We included core tests of cortico-subcortical integrity from the Cambridge Neuropsychological Test Automated Battery (CANTAB), including visual Pattern Recognition Memory, Spatial Working Memory, and the Tower of London (http://www.camcog.com). Given the recent interest in impulsivity in the context of these disorders (Chamberlain et al., 2005), we also included a test of the tendency to gather and evaluate information prior to making a decision ('reflection-impulsivity') (Clark, Robbins, Ersche, & Sahakian, 2006). As decision-making had yet to be assessed in trichotillomania, we also included the Cambridge gamble task, which has been linked to orbitofrontal cortex function (Murphy et al., 2001; Rahman, Sahakian, Hodges, Rogers, & Robbins, 1999; Rogers, Everitt et al., 1999). The probabilistic learning and reversal test was included, which is also thought to be sensitive to orbitofrontal cortex function (Clark, Cools, & Robbins, 2004; Evers et al., 2005; Fellows & Farah, 2003). Finally, the affective go/no-go test was also incorporated into the battery. This test is sensitive to affective processing abnormalities in mood disorders (Erickson et al., 2005; Murphy et al., 1999).

Given that the symptoms of OCD but not trichotillomania suggest problems with high-level cognitive rigidity, it was hypothesized that OCD patients would show cognitive deficits across a broader range of domains than for trichotillomania. In previous work we identified overlapping motor impulsivity, as indexed by the stop-signal paradigm, in these two disorders (Chamberlain et al., 2006b). Therefore, it was hypothesized that OCD and trichotillomania might also share overlap in another type of impulsivity: reflection-impulsivity, which refers to the pre-decisional aspect of behavioural regulation where information is gathered and evaluated prior to making a decision. None of the cognitive tests in the present study had been evaluated in trichotillomania patients previously, to our knowledge. While neurocognitive findings in OCD to date are somewhat mixed, at least one prior study found impaired CANTAB Pattern Recognition Memory versus controls (Watkins et al., 2005), and another found impaired Tower of London performance associated with abnormal fronto-striatal dysfunction (van den Heuvel et al., 2005). Therefore, we predicted impaired Pattern Recognition Memory and Tower of London performance in OCD. The Cambridge gamble task, along with the probabilistic learning task, are thought to be sensitive to orbitofrontal cortex pathology (Clark et al., 2004). While prior studies have not examined probabilistic learning on OCD, intact performance has been reported on Cambridge Gamble versus controls (Watkins et al., 2005). Therefore, we predicted intact Cambridge gamble and probabilistic learning in OCD. Finally, we predicted that neither clinical group would show a similar mood bias to that previously reported in depressed patients on the Affective go/no-go test, as we were careful to exclude patients with co-morbid mood disorders (see below).

2. Methods

The study was approved by the Cambridge Local Research Ethics Committee. Patients were recruited via an outpatient mental health centre pool of approximately 200 patients after being screened by a consultant psychiatrist (NF) specializing in obsessive–compulsive spectrum disorders, using extended clinical interview supplemented with the Mini International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998). Patients met DSM-IV criteria for their respective conditions. The MINI screened out depression/mania, other anxiety disorders, alcohol abuse and dependence, non-alcohol psychoactive substance use disorders, and psychotic disorders. We also excluded patients with Tourette's syndrome, tic-spectrum disorders, ADHD and other impulse control disorders (such as compulsive gambling and compulsive shopping), as part of the extended clinical interview and mental state assessment. Controls were recruited via newspaper advertisements, and were included on the basis of not experiencing current axis-I disorders, and no significant past history of axis-I disorders, according to assessment with the MINI and interview. Exclusion criteria for all participants included scores >16 on the Montgomery-Asberg Depression Rating Scale (MADRS) (Montgomery & Asberg, 1979), or a significant past history of neurological illness or head injury. In the OCD group, symptom dimensions and disease severity were assessed using the Yale-Brown obsessive compulsive scale (Y-BOCS) (minimum total score for study inclusion = 7) (Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989), and only patients with principally washing/checking symptoms without hoarding were included. Within the trichotillomania group, disease severity was assessed using the Massachusetts General Hospital Hair-pulling scale (MGH) (minimum total score for study inclusion = 7) (Keuthen, O'Sullivan, & Sprich-Buckminster, 1998). We were careful to exclude OCD from the trichotillomania group and vice versa, by explicit screening during the interview.

Twenty OCD patients, 20 trichotillomania patients, and 20 controls were recruited into the study after meeting criteria and providing written informed consent. Sixteen OCD participants were receiving stable doses of selective serotonin reuptake inhibitors (SSRIs) at the time of testing. The remaining four OCD patients, and all trichotillomania patients, were free from psychotropic medication for at least 6 months prior to study participation. Verbal IQ was estimated using the National Adult Reading Test (NART) (Nelson, 1982). Impulsive personality traits were assessed using the Barratt Impulsivity Scale (BIS) (Patton, Stanford, & Barratt, 1995). Participants completed a battery of neurocognitive tests taking approximately 2.5 h, in a quiet testing environment with an experienced neuropsychologist. Data for three tasks (response inhibition, set-shifting, and visuo-spatial novel sequence generation) have been reported previously for the majority of these volunteers and are therefore not reported here (Chamberlain et al., 2006b; Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2006a).

2.1. Neuropsychological testing battery

Brief descriptions for each neuropsychological test are provided below. Tests were fully counter-balanced within groups as appropriate.

2.1.1. Pattern recognition memory (Sahakian et al., 1988)

The participant is asked to memorise several abstract patterns presented sequentially on the computer screen. After viewing each stimulus set (of 12 stimuli), the stimuli are presented a second time, each paired with a novel distractor. The participant must identify the familiar pattern using a touch-screen. Feedback (correct or incorrect) is given after each choice. The recognition phase is presented a second time after approximately 25 min to assess delayed pattern recognition memory. Dependent variables are the percentage of patterns correctly recognised for the immediate and delayed recognition conditions.

2.1.2. Spatial working memory (Owen, Downes, Sahakian, Polkey, & Robbins, 1990)

This is a self-ordered search task where the participant searches an array of boxes for hidden tokens. On any given search, there is a single token available, and the token will never appear in a box that has already yielded a token. Thus, the subject can progressively narrow their search to boxes that have not previously yielded tokens. The trial is completed when a token has been found in each box. The number of search locations (boxes) increases across the task, with inclusion of both easy (4 and 6 boxes) and hard (8 and 12 boxes) problems. Dependent variables are the total number of between-search errors (inappropriately returning to boxes where tokens were previously found) for easy and hard levels of difficulty; and strategy scores—with lower scores representing superior use of self-ordered strategies to optimize performance.

2.1.3. Tower of London (Owen et al., 1990)

The Tower of London task was derived from the classic test of executive planning. The participant must calculate the number of moves required to match two arrangements of balls. Subjects begin the task with a practice stage on the CANTAB Stockings of Cambridge task, where they must actually move balls in the lower arrangement, using the touch-screen. During the 'one-touch' test phase, problem difficulty (the actual number of moves) varies from 1 to 6. Dependent variables are the mean number of attempts made prior to obtaining the correct solution for easy (1–3 moves) and hard (4–6 moves) levels of difficulty.

2.1.4. Information sampling task (Clark et al., 2006)

This test examines the tendency to gather and evaluate information prior to making a decision, and assesses a similar underlying process ('reflection-impulsivity') to the Matching Familiar Figures Test (Kagan, 1966). The participant views a grid of 25 closed boxes, which can be opened one at a time by touching the screen, to reveal an underlying distribution of two colours (e.g. yellow or brown). The participant is asked to decide, on each trial, which of the two colours is in the majority. They are instructed that they can open as many boxes as they wish in order to reach their decision. When they have reached their decision, they indicate their response by touching a coloured square at the foot of the screen. Participants perform 10 trials under each of two points conditions: in the fixed reward condition, the participant will win 100 points for a correct response regardless of the number of boxes opened. In the decrementing reward condition, the number of points the volunteer stands to win decreases by 10 for each box uncovered, to introduce a conflict between level of certainty and the available win. Incorrect responses yield a 100-point penalty in both conditions. The key measures for this task are the mean number of boxes opened in each of the two conditions.

2.1.5. Affective go/no-go (Murphy et al., 1999)

The affective go/no-go task examines mood-processing bias. Positive- and negatively valenced words are presented rapidly on the computer screen. Within a given block, one valence is labeled 'targets' and the participant must respond to targets by pressing the space bar. The other valence is labeled 'distractors' and the participants avoid making motor responses to distractor words. There are 10 blocks in total, where target valence alternates in an AABBAA design. Valence in the first block was counter-balanced within groups. Key variables are the affective reaction time bias (overall difference in response times between sad and happy words), commission errors for happy and sad distractors, and omission errors for happy and sad targets. This task is sensitive to abnormal affective processing bias in depression and mania mediated by mood-cognition interactions sub-served by the orbitofrontal cortex and associated regions (Murphy et al., 1999).

2.1.6. Cambridge gamble task (Rogers, Owen et al., 1999)

On the Cambridge gamble (decision-making) task, volunteers attempt to accumulate as many points as possible by gambling over a range of probabilities of winning. For each trial, 10 boxes are shown on-screen and volunteers are told that a token is hidden behind either a red or blue box. The proportion of red and blue boxes is varied over the course of the task—for example, a ratio of 1:9 red:blue boxes would indicate a 90% chance of the token being hidden behind a blue box. After examining the proportion of coloured boxes, the participant indicates which of the two colours s/he thinks the token is hidden behind, and then has to make a decision as to how many points to gamble that this decision is correct. The amount gambled on each trial is determined by the volunteer hitting a points counter when it reaches a level that they are happy to bet at. The counter either increments (5%, 25%, 50%, 75%, 95% of total collected points) or decrements (reverse order) over time. Key outcome measures include the mean percentage of points gambled, and the quality of decision-making (percentage of rational decisions made). Decision-making tasks such as these have been argued to represent sensitive measures of orbitofrontal cortex pathology in psychiatric disorders (Clark et al., 2004).

2.1.7. Probabilistic learning and reversal (Swainson et al., 2000)

The probabilistic learning and reversal task examines the ability to acquire and reverse a two-choice visual discrimination. Choices yield probabilistic feedback in an 80:20 ratio, such that 20% of correct responses yield incorrect feedback. Subjects complete 40 acquisition trials and 40 trials after reversal of the rule. The probabilistic feedback introduces a degree of ambiguity when the reversal occurs, and encourages perseveration to the previously reinforced stimulus. Key variables are the proportion of subjects reaching a criterion of eight consecutive correct responses and number of errors made prior to reaching criterion for each of the two task stages, and the number of perseverations made when the rule change occurs. Ability to reverse stimulus-reward contingencies has been reported to be dependent on orbitofrontal cortex but not dorsolateral prefrontal cortex in animal models and in human lesion patients (Berlin, Rolls, & Kischka, 2004; Clarke, Dalley, Crofts, Robbins, & Roberts, 2004; Clarke et al., 2005).

2.2. Statistical analyses

Data were examined using one-way analysis of variance (ANOVA) as indicated. In order to control for the possibility of false positives, significance was set at 0.05 divided by the number of dependent variables within each test. Where groups differed significantly on the ANOVA, least significant difference (L.S.D.) tests were then undertaken to compare: (i) OCD versus controls; (ii) trichotillomania versus controls; (iii) OCD versus trichotillomania. Significance threshold for pair-wise comparisons was set a priori at $p < 0.05$. For significant pair-wise differences, effect sizes were calculated (Cohen's D). For variables where nor-

mality was violated, data were transformed to improve suitability for parametric statistics (Cardinal & Aitken, 2006). For tasks yielding significant group differences overall, correlation analyses (Pearson's *r*) were undertaken between these measures and Y-BOCS total scores (OCD group), MGH total score (trichotillomania group), and total MADRS scores (within each group and then with all data pooled).

3. Results

3.1. Demographic and clinical characteristics

As can be seen in Table 1, groups did not differ significantly in the ANOVA for age, education, verbal IQ, and mood scores. Total impulsive personality trait scores according to the BIS did not differ significantly between groups. Groups did not differ in terms of male:female ratio (chi-squared analysis). Mean Y-BOCS score in the OCD group was 20.40 (±S.D. 4.07), representing mild–moderate disease severity. In the trichotillomania group, mean MGH score was 13.95 (±4.50), representing mild–moderate disease severity. Neurocognitive findings for each task are described in detail below, and are presented in summary form in Table 2.

3.1.1. Pattern recognition memory

Separate ANOVAs were conducted for immediate and delayed recognition conditions (significance threshold $p < 0.025$). Groups differed significantly in the immediate [$F(2,57) = 4.882$, $p = 0.012$] but not delayed [$F(2,57) = 2.423$, $p = 0.098$] recognition conditions. Post hoc tests revealed that the OCD patients were impaired relative to both other groups at the immediate recognition stage ($p < 0.05$, Table 2) whereas trichotillomania patients did not differ significantly from controls.

3.1.2. Spatial working memory

Data for total between-search errors at easy and hard difficulty levels, and for overall strategy scores, were entered into separate ANOVAs (significance threshold $p < 0.017$). Groups differed significantly on the hard [$F(2,57) = 4.995$, $p = 0.010$] but not easy [$F(2,57) = 0.362$, $p > 0.30$] levels of difficulty for between-search errors. Post hoc tests (Table 2) revealed that OCD and trichotillomania patients made significantly more errors than controls at the harder levels of difficulty. Groups did not differ on strategy scores in a one-way ANOVA [$F(2,57) = 1.535$, $p > 0.20$].

3.1.3. Tower of London

Data were entered into two separate ANOVAs for easy and hard problems (significance threshold $p < 0.025$). Groups differed significantly at the hard [$F(2,57) = 4.883$, $p = 0.012$] but not at the easy [$F(2,57) = 0.737$, $p > 0.30$] levels of difficulty. Follow-up tests (Table 2) revealed that the OCD group required more attempts to obtain the correct response overall compared to both control and trichotillomania groups at the hard levels of difficulty ($p < 0.05$). Performance of trichotillomania and controls did not differ significantly.

3.1.4. Information sampling task

Data were analysed using two separate one-way ANOVAs (significance threshold $p < 0.025$). There was some evidence that groups differed in terms of the mean number of boxes opened in the fixed reward condition [$F(2,57) = 3.367$, $p = 0.041$], but this did not meet the significance threshold after corrections for multiple comparisons. Groups did not differ significantly in terms of the number of boxes opened in the decrementing reward condition [$F(2,57) = 2.516$, $p = 0.090$]. In order to examine whether individual groups altered their information sampling behaviour between fixed reward and decrementing reward conditions, paired *t*-tests were conducted. There was a significant paired difference within the trichotillomania group [$t(19) = 3.202$, $p = 0.005$] and control group [$t(19) = 5.139$, $p < 0.001$]—subjects in these groups adapted their information sampling behaviour in accordance with the change in reinforcement contingencies. However, the paired *t*-test between conditions did not reach significance in the OCD group [$t(19) = 1.966$, $p = 0.064$].

3.1.5. Affective go/no-go

ANOVAs were conducted to compare groups on overall reaction time bias towards happy words, and errors for each block category (happy/sad targets/distractors, Table 2) (significance threshold $p < 0.01$). Groups did not differ for happy affective reaction time bias [$F(2,57) = 0.333$, $p = 0.718$] nor for the number of commission errors for either happy [$F(2,57) = 2.104$, $p = 0.131$] or sad [$F(2,57) = 2.723$, $p = 0.074$] distractor words. Groups did differ in terms of the number of omission errors to sad [$F(2,57) = 6.410$, $p < 0.01$] but not to happy target words [$F(2,57) = 1.564$, $p > 0.10$]. This was attributable to OCD patients making significantly more omission errors to sad words compared to both other groups [$p < 0.05$] (Table 2). Trichotillomania and controls did not differ on this measure.

Table 1
Demographic and clinical characteristics of the sample

	OCD patients		TTM patients		Controls		ANOVA (d.f. = 2, 57)	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	*F*	Significance
Age (years)	35.3	14.07	36	12.39	32.15	7.7	0.614	n.s.
Verbal IQ	115.72	5.86	118.35	7.1	117.34	5.59	0.909	n.s.
Education	2.8	1.01	2.8	0.77	2.95	0.76	0.207	n.s.
Barratt	64	8.12	67.7	9.4	64.5	11.58	0.838	n.s.
MADRS	6.85	4.42	4.6	3.95	3.7	4.56	2.816	n.s.

MADRS, Montgomery-Asberg Depression Rating Scale (Montgomery & Asberg, 1979).

Table 2
Neuropsychological test results

Task and measure	OCD patients		TTM patients		Controls		ANOVA	Pairwise comparisons (L.S.D. tests)			Effect size (Cohen's *D*)		
	Mean	S.D.	Mean	S.D.	Mean	S.D.		OCD vs. C	TTM vs. C	OCD vs. TTM	OCD vs. C	TTM vs. C	OCD vs. TTM
Pattern recognition													
% immediate recognition	86.25	9.94	93	7.78	93.5	6.69	Sig.	*	n.s.	*	0.86		0.75
% delayed recognition	78.33	13.2	84.8	12.1	86	10.2	n.s.						
Spatial working memory													
Between-search errors, easy	6.7	7.77	5.85	8.16	4.8	4.82	n.s.						
Between-search errors, hard	43.55	17.4	48	13.5	33.1	14.8	Sig.	*	*	n.s.	0.65	1.05	
Strategy scores (lower = better)	36.45	6	36	8.44	32.5	8.69	n.s.						
Tower of London													
Attempts to correct solution, easy	1.09	0.12	1.05	0.09	1.08	0.07	n.s						
Attempts to correct solution, hard	1.84	0.53	1.5	0.26	1.5	0.34	Sig.	*	n.s.	*	0.76		0.81
Information gathering (reflection-impulsivity)													
Boxes opened, fixed reward	10.61	4.26	10.7	4.32	14.1	5.94	n.s.						
Boxes opened, decrementing reward	9.18	2.79	8.35	2.64	7.47	3.02	n.s.						
Affective go/no-go													
Happy affective bias (ms)	8.67	36.5	15.8	33.1	9	21.6	n.s.						
Mean commission errors													
Happy distractors	1.52	0.98	1.46	0.78	1.16	0.5	n.s.						
Sad distractors	1.58	1.02	1.45	0.68	1.12	0.52	n.s.						
Mean omission errors													
Happy targets	0.87	0.76	0.76	0.65	0.51	0.55	n.s.						
Sad targets	1.38	0.99	0.53	0.57	0.76	0.71	Sig.	*	n.s.	*	0.72		1.05
Cambridge gamble													
% points gambled	56.05	16.2	55.5	15.5	60.1	13.5	n.s.						
% rational decisions	98.00	4.00	97.00	6.00	99.00	2.00	n.s.						
Probabilistic learning													
Stage 1													
Proportion passing#	0.85		0.9		1		n.s.						
Errors to criterion	1.55	2.54	1.25	1.86	0.4	0.94	n.s.						
Stage 2													
Proportion passing#	0.9		0.9		0.9		n.s.						
Errors to criterion	5.1	2.2	4.55	1.54	4.15	1.9	n.s.						
Perseverations	3.5	2.7	3.5	1.76	3.2	1.77	n.s.						

Sig.: significant in one-way ANOVA at designated *p* cut-off (0.05/number of test measures), see text pair-wise comparisons were undertaken only where significant group differences were detected on ANOVA. *$p < 0.05$, **$p < 0.01$ #chi-squared analysis d.f. = 2.

3.1.6. Cambridge gamble

Data for the overall percentage of points gambled and for the percentage of rational decisions made were entered into two one-way ANOVAs (significance threshold $p < 0.025$). Groups did not differ on either measure [$F(2,57) = 1.431$, $p = 0.247$; $F(2,57) = 1.350$, $p = 0.268$].

3.1.7. Probabilistic learning and reversal

Data for the proportion of people passing each of the two stages, the number of errors made prior to attaining criteria for each stage, and number of perseverative errors were entered into separate one-way ANOVAs (significance threshold $p < 0.01$). There was no effect of group on the number of errors made in either stage of the task [stage 1: $F(2,57) = 1.971$, $p > 0.10$; stage 2: $F(2,57) = 1.263$, $p > 0.20$], nor on perseveration-type errors after reversal [$F(2,57) = 0.133$, $p > 0.30$]. Groups did not differ in terms of the proportion of subjects reaching criterion for each stage in the chi-squared analysis (Table 2).

3.1.8. Correlation analyses

For tests yielding group differences in the ANOVA analyses, correlation analyses were undertaken between these measures

and: Y-BOCS scores within OCD group, MGH scores within trichotillomania group, and MADRS scores (within each group, and with all data pooled). None of these correlations approached significance (all $p > 0.20$).

4. Discussion

To our knowledge, this is the first study to compare a broad range of neuropsychological functions in OCD and trichotillomania patients who were free from axis-I co-morbidities. These two disorders share overlapping phenomenology (repetitive inappropriate motor behaviour), and familial transmission (Fontenelle, Mendlowicz, & Versiani, 2005); Lenane et al., 1992; Lochner et al., 2005; Stanley et al., 1997; Swedo & Leonard, 1992). OCD is associated with abnormalities in fronto-striatal circuitry especially orbitofrontal cortex, anterior cingulate cortex, and basal ganglia (caudate) (Graybiel & Rauch, 2000; Nielen & Den Boer, 2003; van den Heuvel et al., 2005), while the extent of neural involvement in trichotillomania is somewhat unclear due to a paucity of research. Caution is required when drawing inferences regarding likely neural abnormalities on the basis of behavioural data. Nonetheless, the finding of a more restricted profile of cognitive dysfunction in trichotillomania compared to OCD may suggest more focal involvement of elements of fronto-striatal circuitry. This issue should now be addressed in follow-up structural and functional neuroimaging investigations.

On the CANTAB Spatial Working Memory task, OCD and trichotillomania patients showed increased numbers of between-search errors at the harder levels of difficulty in the absence of impaired strategy scores. In prior work, patients with frontal lesions showed impaired strategy and increased errors whereas patients with Parkinson's disease showed increased errors only (Owen et al., 1993; Owen, Morris, Sahakian, Polkey, & Robbins, 1996). Therefore, these findings may implicate basal ganglia dysfunction in OCD and trichotillomania. While prior studies have found intact performance of OCD patients on versions of the CANTAB Spatial Working Memory task, we employed a more difficult paradigm with up to twelve search locations, which likely increased sensitivity to fronto-striatal dysfunction. We believe this to be the first time self-ordered spatial working memory has been examined in trichotillomania. Overlapping impairments in spatial memory have been reported for OCD and trichotillomania using a very different spatial memory task – the Stylus Maze test – in a prior study (Rettew, Cheslow, & Rapoport, 1991).

It has been proposed that OCD, trichotillomania, and other disorders may be conceptualized as part of a 'spectrum' or 'family' of obsessive–compulsive disorders associated with inhibitory control deficits manifesting as excessive motoric output (Chamberlain et al., 2005). We reported previously that OCD and trichotillomania patients showed overlapping deficits in an aspect of impulsivity relating to 'motor inhibition' or the tendency to suppress pre-potent motor responses (stop-signal test) (Chamberlain et al., 2006b). However, impulsivity is not a unitary construct in psychiatry (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). In the present study, groups did not differ significantly in terms of the tendency to gather and evaluate information, as indexed by number of boxes opened, on a recently developed test of reflection-impulsivity, which is sensitive to substance abuse (Clark et al., 2006). The performance of OCD patients did differentiate from that of trichotillomania patients on another aspect of this test. In contrast to the control and trichotillomania groups, the OCD subjects did not significantly alter their information sampling behaviour between the fixed and decrementing reward conditions ($p = 0.064$). That is, when an incentive was introduced for low-certainty responding, healthy controls and trichotillomania subjects were sensitive to this change in task structure and opened fewer boxes in the decrementing reward condition (both $p < 0.01$). The OCD group in contrast maintained more similar information sampling between conditions which may be a further manifestation of cognitive rigidity in OCD to that previously reported on tests of strategy and attentional flexibility (Chamberlain et al., 2006b; Chamberlain, Blackwell, et al., 2006a).

Only OCD patients were impaired on the one-touch Tower of London task, a test of executive planning in which volunteers use forward planning to calculate the number of moves needed to match a goal arrangement shown by the computer (Owen et al., 1990). Some previous studies have found intact Tower of London performance in OCD as indexed by accuracy of responses (Veale, Sahakian, Owen, & Marks,1996; Watkins et al., 2005). However, we employed a more difficult 'one-touch' version of the task that required volunteers to work out problems in-mind (rather than physically on-screen) and that incremented to six-move problems, and these features are likely to maximize test sensitivity. Indeed, deficits in the OCD group were found only at the harder levels of the task. Abnormalities in fronto-striatal circuitry have also been reported in OCD patients using a functional magnetic resonance imaging (fMRI) version of this task (van den Heuvel et al., 2005).

OCD patients, but not trichotillomania patients, were also impaired on visual pattern recognition memory (immediate recognition) consistent with a previous study comparing OCD patients against controls (Watkins et al., 2005). While the OCD patients also showed a tendency to perform more poorly than controls and trichotillomania patients on the delayed recognition stage of this task, this difference did not reach statistical significance. OCD patients also differed from trichotillomania and controls on a test of affective processing (affective go/no-go test), in that they missed more responses to target sad words than other groups (omission errors). Rather than indicating a depressive bias, this result suggests a bias *against* responding to sad words. It is noteworthy that most OCD patients were medicated on SSRIs, and that such medications have been shown to modulate affective processing away from negative information (Harmer, Hill, Taylor, Cowen, & Goodwin, 2003; Harmer, Shelley, Cowen, & Goodwin, 2004). However, there was no evidence for abnormal affective reaction time bias in the OCD group.

It has been suggested that OCD may be characterised as a disorder of decision-making (Sachdev & Malhi, 2005), and that OCD and trichotillomania symptoms may lie on a spectrum polarised at one end by risk-aversion and at the other by

extreme risk-seeking (Hollander & Cohen, 1996). There was no evidence for abnormalities in decision-making (whether risk aversive or risk-seeking) on the Cambridge gamble task in either clinical group. In a prior study using this test, intact decision-making was also found in OCD compared to healthy controls (Watkins et al., 2005). Using a different test of decision-making, the Bechara/Iowa gambling task, one study found intact performance in OCD versus controls (Nielen, Veltman, de Jong, Mulder, & den Boer, 2002) whereas another found impaired performance; especially, in OCD patients who did not respond to SSRI-treatment (Cavedini et al., 2002). To our knowledge, decision-making has not previously been examined in trichotillomania. Performance on the Cambridge gamble task, along with the probabilistic learning task, is thought to depend upon the integrity of the orbitofrontal cortex (Fellows & Farah, 2003; Hornak et al., 2003; Rogers, Everitt et al., 1999; Rolls, Hornak, Wade, & McGrath, 1994) (but see also, Manes et al., 2002). Intact performance of OCD patients on these two tasks supports the neuroanatomical model proposing that hyperactivation in this region in OCD patients (observed in neuroimaging studies) may not represent orbitofrontal pathology *per se*, but reflects compensatory responses for neuropathological deficits 'downstream' at other fronto-striatal stations (Graybiel & Rauch, 2000; Saxena, Brody, Schwartz, & Baxter, 1998). Alternatively, SSRI treatment might have exerted a modulatory effect on orbitofrontal cortical function in our sample, as 16/20 OCD patients were stabilised on SSRI medication at the time of testing. There is evidence that acute serotonin manipulations can modulate cognitive tests dependent upon the orbitofrontal cortex in healthy volunteers (Chamberlain, Muller, et al., 2006; Rogers, Blackshaw et al., 1999; Rogers et al., 2003), and that chronic SSRI-treatment modulates orbitofrontal cortex activity in people with OCD, e.g. Nakao et al. (2005) and Saxena et al. (1999).

A number of caveats should be noted. As reported above, the majority of OCD patients in the present study were stabilized on SSRI medication at the time of testing whereas the trichotillomania patients were medication-free and it is possible that this may have differentially contributed to the findings. However, most studies to date suggest that OCD patients receiving chronic SSRI treatment show cognitive deficits that persist despite treatment but do not worsen (Borkowska, Pilaczynska, Araszkiewicz, & Rybakowski, 2002; Fontenelle et al., 2006; Mataix-Cols, Alonso, Pifarre, Menchon, & Vallejo, 2002; Nielen & Den Boer, 2003; Roh et al., 2005). Furthermore, cognitive deficits comparable to those in OCD patients have been reported in medication-naïve first-degree relatives of such patients across some cognitive domains (Chamberlain, Blackwell, et al., in press-a). Nonetheless, this does leave open the important question as to what action SSRIs have on cognitive function in OCD. Groups did not differ overall for total depressive mood (MADRS) scores in the ANOVA, but there was a trend towards a group difference, and OCD patients showed marginally higher scores on the MADRS than controls. It is unlikely that depression contributed to the findings in the OCD group as: (i) no correlations were found between MADRS scores and cognitive functions; (ii) patients were included on the basis of being free from depression according to DSM-IV criteria and MADRS cut-off; (iii) mean MADRS scores were well-beneath cut-off even for depression in full remission; (iv) MADRS includes items such as difficulties with concentration which would be expected to arise consequential to OCD symptoms *per se*; (v) the pattern of abnormality on the affective go/no-go task was not the same as that reported in depression. It is important to note that, as we carefully selected patients without co-morbidities who were generally of mild–moderate disease severity, this may limit the generalisability of these findings to other patient subgroups. For example, there is growing evidence for an early-onset form of OCD associated with co-morbid anxiety disorders and more severe symptoms (Fontenelle et al., 2005; Matsunaga et al., 2005), and it would be of interest to examine the neurocognitive performance of such a subgroup.

In sum, this study has provided important evidence for overlapping but also differential cognitive deficits in OCD and trichotillomania. Future studies should investigate cognitive functions in larger patient samples, and assess the structural and functional neuroimaging correlates of these abnormalities. In particular, it will be important to address the relationship between cognitive dysfunction and the expression of clinical phenotypes—i.e. whether the deficits reported herein reflect trait (candidate endophenotype) or state (directly associated with symptoms) abnormalities.

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