Neuropsychiatric features of punding and hobbyism in Parkinson's disease

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ABSTRACT

Background. Little is known about the cognitive and neuropsychiatric profile associated with punding and hobbyism in Parkinson's disease (PD). **Objective.** To compare the clinical and neuropsychological features of PD patients with punding and hobbyism to PD controls. **Methods.** The Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease – Rating Scale (QUIP-RS) was used as a screening tool, and a structured interview was used to diagnose punding/hobbyism. Clinical and neuropsychological assessment was conducted with validated questionnaires/scales. **Results.** Twenty-one patients with PD and punding (PD+pu) were compared to 26 with hobbyism (PD+h) and 25 PD controls. PD+pu patients showed higher levels of anxiety, non-motor symptoms and motor symptoms, and lower Frontal Assessment Battery scores. The PD+h group exhibited similar levels of anxiety and motor fluctuations to the PD+pu group. **Conclusion.** PD+pu showed increased anxiety and frontal lobe dysfunction, similar to PD+h. Hobbyism could be a prodromal phase with increased risk of leading to punding.

Punding is a stereotyped non-goal orientated behaviour, characterized by repetitive manipulations of technical equipment, the continual handling, examining, and sorting of common objects, grooming, hoarding, pointless driving or aimless walkabouts.[1] Initially described among amphetamine and cocaine users, punding was first reported in an l-dopa treated patient with Parkinson's disease (PD) by Friedman in 1994.[2] The prevalence of reported punding in PD varies between 1.4%[3] up to 14%.[4] While enjoying a hobby is a healthy habit, when there is frequent preoccupation in pursuing the activity resulting in negative consequences to an individual's personal life it can represent excessive hobbyism, a form of behavioural addiction.

There is an idiosyncratic quality to punding, with previous occupations and pre-morbid hobbies and pastimes influencing the type of abnormal behaviour.[3] Patients with punding have more psychiatric symptoms, greater impulsivity and are more likely to have other impulsive compulsive behaviours (ICBs).[4] Punding has also been linked with the dopamine dysregulation syndrome[3] (Lees syndrome[5]) and l-dopa peak dose dyskinesias.[6] Although excessive hobbyism is commonly associated with punding and placed together with punding in the QUIP-RS, the relationship between these two behavioural abnormalities has not been fully elucidated yet. Hobbyism and punding could represent a spectrum of the same behaviour. In this study, we compared the clinical and neuropsychiatric features of PD patients with punding and excessive hobbyism to PD patients without punding and other ICBs.

Methods

PD patients without punding or other ICBs were recruited from a movement disorders outpatient clinic at the National Hospital for Neurology and Neurosurgery, Queen Square, London, UK.

A diagnosis of punding or hobbyism was made based on the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease – Rating Scale (QUIP-RS) using previously published cut-off scores[7] and confirmed with a structured interview. PD controls also completed the QUIP-RS and structured interview to confirm the absence

of punding or other ICBs. The main features elucidated on interview to distinguish between punding and hobbyism relates to whether the behaviour causes occupational and/or social dysfunction (punding) or not (hobbyism), as determined by the treating clinician, following review with the patient themselves, and their carers. Participants self-completed the following questionnaires: the Hospital Anxiety and Depression Scale (HADS), the 36-Item Short Form Survey (SF36), the Barratt Impulsiveness Scale (BIS11), the REM Sleep Behaviour Disorder Screening Questionnaire (RBDq) and the Apathy Scale (AS). A Unified Parkinson's Disease Rating Scale (UPDRS) parts I and III, the Abnormal Involuntary Movement Scale (AIMS), the Frontal Assessment Battery (FAB), the Montreal Cognitive Assessment (MoCA), and the Stroop test (EncephalApp Stroop Test[8]) were completed by the investigating physician.

The study received approval by the Queen Square Ethics Committee (15.LO.1531) and all participants gave informed consent. All variables were tested for normality and statistical tests were chosen accordingly. Proportions were compared with the Pearson chi-square test, provided the minimum expected cell count was more than five. Data was analysed using SPSS©22.

Results

Forty-seven patients screened positive for punding/hobbyism in the QUIP-RS: 21 were diagnosed with punding as the dominant behavioural phenotype (PD+pu) and 26 with excessive hobbyism without pathological punding (PD+h) after the structured interview. 25 PD control patients without hobbyism, punding or other ICBs were matched for age, duration of illness and medication use (Table 1).

Table 1 shows the neuropsychiatric and cognitive characteristics of the three groups. Differences were seen in the HADS anxiety subscale and total score, UPDRS parts I and III, and FAB scores. None of the SF36 subscales differed between PD+pu, PD+h and controls. Patients with punding showed lower (worse) FAB scores, whereas the MoCA scores difference did not reach significance.

Pairwise post hoc comparisons showed that, compared to controls, individuals with punding exhibited higher scores on the HADS total score and anxiety subscale, higher scores on the UPDRS parts I and III and lower (worse) scores on the FAB. The only difference seen in the comparison between PD+pu and PD+h was a higher burden of motor symptoms and non-significant lower FAB scores among individuals with punding (see supplementary materials). Among patients with excessive hobbyism: 26/16 had comorbid ICBs (61,5%). Among patients with punding: 21/12 had comorbid ICBs (57%).

Table 1 – Demographic and clinical characteristics

	Punding	Hobbyism	PD controls	Test statistic
N	21	26	25	
Males (%)	15 (71.4%)	23 (88.5%)	18 (72%)	$x^2(2)=2.690$; p=0.261
Age	59.5 (± 16)	56.0 (± 11.7)	59.8 (± 9)	H(2)=6.549; p=0.055
Age at PD onset (years)	47.6 (± 9.7)	42.3 (± 10.6)	47.56 (± 7.9)	F(2)=0.728; p=0.486
Disease duration (years)	15.2 (± 6.8)	13.8 (± 9.7)	12.4 (± 6.7)	H(2)=1.832; p=0.400
Dyskinesias (%)	16 (80%)	21 (80.8%)	15 (60%)	$x^2(2)=3.454$; p=0.178
Motor fluctuations (%)	20 (95.2%)	23 (88.4%)	18 (72%)	$x^2(2)=5.201$; p=0.074
DBS (%)	4 (19%)	1 (3.8%)	4 (16%)	$x^2(2)=2.883$; p=0.237
Dopamine Agonist (DA)(%)	9 (42.9%)	13 (50%)	13 (52%)	$x^2(2)=0.413$; p=0.813
Amantadine use (%)	15 (71.4%)	15 (57.7%)	12 (48%)	$x^2(2)=2.584$; p=0.275
MAOI use (%)	6 (28.6%)	3 (11.5%)	3 (12%)	$x^2(2)=3.027$; p=0.220
L-dopa equivalent daily dose (LEDD)[9]	1092 (± 333)	1089 (± 495)	893.8 (± 526)	F(2)=1.457; p=0.240

DA dose in LEDD	227 (± 165) N=9	175 (±138) N=13	257 (±127) N=13	H(2)=2.916; p=0.233
HADS				
Total	18.2 (± 6)	16 (± 5.8)	13.1 (± 6)	**F(2)=3.727; p=0.029**
Anxiety	10.4 (± 3.9)	8.5 (± 3.7)	6.8 (± 4)	**F(2)=4.631; p=0.013**
Depression	7.8 (± 3.9)	7.5 (± 3.3)	6.6 (± 3)	F(2)=0.642; p=0.529
UPDRS part I	21.6 (± 9.9) N=20	17.7 (± 7.4) N=24	13.2 (± 5) N=18	**F(2)=5.433; p=0.007**
UPDRS part III	34.4 (± 12.9)	24.6 (± 8.1)	24.4 (± 14)	**F(2)=5.184; p=0.008**
BIS11	67.8 (± 8) N=17	67.8 (± 6) N= 25	64.1 (± 6) N= 22	F(2)=1.990; p=0.145
AIMS	7.7 (± 7)	7 (± 5)	4.8 (± 6)	H(2)=3.282; p=0.194
FAB	14.7 (± 3)	16.3 (± 1)	16.6 (± 2)	**H(2)=7.634; p=0.022**
MoCA	24.3 (± 6)	26.8 (± 3)	27.1 (± 2)	H(2)=2.406; p=0.300
AS	15 (± 5) N=18	14 (± 7) N=22	13 (± 5) N=18	H(2)=1.399; p=0.497
Stroop reaction time (s)	20.8 (± 7) N=6	18.5 (± 4) N=15	16.34 (± 2.3) N=12	F(2)=2.185; p=0.130
Stroop errors	1.4 (± 2.5)	1.25 (± 1.5)	0.42 (± 0.5)	H(2)=1.069; p=0.497
			33.2 (± 16)	
SF36 general health	39.9 (± 24) N=20	40.9 (± 18)	33.2 (± 16)	H(2)=1.665; p=0.435

PD+pu – patients with Parkinson's disease and punding; PD-pu: patients with Parkinson's disease without punding; PD – Parkinson's disease; DBS – deep brain stimulation; DA – dopamine agonists; MAOI – monoamine oxidase inhibitors; LEDD – l-dopa equivalent daily dose; HADS – Hospital Anxiety and Depression Scale; UPDRS – Unified Parkinson's Disease Rating Scale; BIS11 – Barratt

Impulsiveness Scale; AIMS - Abnormal Involuntary Movement Scale; FAB - Frontal Assessment Battery; MoCA - Montreal Cognitive Assessment; RBD – Rem Sleep Behaviour Disorder; AS – Apathy Scale. Significant results in bold.

Discussion

Punding was associated with increased anxiety, motor and non-motor symptoms and frontal lobe abnormalities on neuropsychological testing. The higher anxiety scores are consistent with one previous study in PD patients with ICBs,[9] and, in line with another study, the depression scores did not differ between groups.[10] There is a complex interplay between dopamine and other neurotransmitters that modulate the appearance of anxiety-like behaviour, however the excessive stimulation of dopaminergic D1/D2 receptors of the mesolimbic, mesocortical and nigrostriatal systems could be driving the increased anxiety seen in the PD+pu group.[11]

Punding is more common in males who develop PD at an earlier age,[12] compatible with our findings. There were no differences in quality of life between groups, contradicting previous data.[13] Considering that the SF36 is a self-assessment questionnaire it is possible that patients with punding failed to report difficulties due to lack of insight. In most cases with punding, the patients' carers are more aware than the patient of the negative impact their excessive behaviours are having on their activities of daily living. Another possible explanation is that the SF36 is less sensitive to changes in PD patients as it has been designed as a general quality of life questionnaire.

Supporting existent evidence for frontal lobe dysfunction, patients with punding scored lower than controls on the FAB. This is also consistent with a recent larger Parkinson's Progression Marker Initiative (PPMI) study which demonstrated attentional dysfunction,[14] and another reporting poorer performance on the Stroop colour naming task associated with thinning of the prefrontal cortex in PD patients with punding.[15] Furthermore, punding also occurs among individuals addicted to cocaine and methamphetamine,[16] conditions that have been associated with frontal

dysfunction,[17] and prefrontal cortical thinning on neuroimaging has been described in PD patients with punding.[15,18] Considering that the selection of motor programs by the basal ganglia is under prefrontal cortex control through top-down fronto-striatal connections,[19] frontal dysfunction could allow stereotypic behaviours associated with excessive dopaminergic stimulation to evade cortical control.

The pathophysiology of the various types of ICBs appears to differ. Over stimulation of dopaminergic D3 receptors associated with the use of DA are the main risk factors for impulse control disorders, such as hypersexuality, pathological gambling, compulsive shopping and eating. Behaviours that fall on the compulsive spectrum, such as dopamine dysregulation syndrome and punding, have been consistently associated with higher doses of dopaminergic therapy.[3,12] Punding behaviour is more commonly seen in association with drugs that stimulate dopaminergic D1 and D2 receptors[10], such as l-dopa. It is not clear whether the use of DA influences the development of punding, with different groups publishing contradictory findings.[4,6] We did not find higher doses of DA in the PD+pu group, suggesting that dopaminergic D3 receptor stimulation associated with the use of DA is not the main mechanism underlying punding.

A higher burden of motor and non-motor symptoms in patients with punding is reported, which could represent higher PD severity. Since PD pathology affects firstly the dorsal striatum, dopaminergic therapy could lead to ICBs by excessive stimulation of the relatively preserved ventral striatum, as suggested in a postmortem study.[20] It is beyond the scope of this paper to assess Lewy body pathology, but theoretically, higher levels of dorsal striatum degeneration could motivate higher intake of PD medication by patients which could, in turn, lead to punding behaviour by excessive dopaminergic stimulation of the more preserved mesolimbic pathway. The appearance of punding behaviour in animals with preserved nigrostriatal systems and in humans without PD following exposure to drugs of abuse [10] favours this hypothesis. Contradictory findings on the burden of PD symptoms in punding has been published,[21,22] and this finding needs to be replicated by future studies.

Patients with excessive hobbyism without pathological punding showed a similar clinical and neuropsychological profile to patients with punding. It is likely that excessive hobbyism in PD is a prodromal phase of punding and patients with this behavioural abnormality should be watched closely for the development of pathological punding. Furthermore, some of the PD+h exhibit definite ICBs, supporting the idea that individuals with excessive hobbyism could be on their way to developing more serious punding behaviour and/or other impulsive compulsive behaviours.

Our sample size is relatively small. This is because we used stringent criteria and only included PD patients who had either no evidence of punding or any other ICBs or had punding or hobbyism as the dominant phenotype. Another limitation is the presence of comorbid ICBs in patients with punding and hobbyism, which could have affected our findings. Nonetheless, this is one of the largest studies looking into cognitive and neuropsychiatric features of punding and excessive hobbyism in PD.

We have found increased anxiety and impaired frontal function in patients with punding. A similar clinical and neuropsychological profile was seen in patients with excessive hobbyism without pathological punding, suggesting that this population is at higher risk of developing punding. Higher stimulation of striatal dopaminergic D1/D2 receptors in individuals with reduced fronto-striatal inhibition could be the mechanism behind punding behaviour.

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Author's roles

PB: conception and design of the study, acquisition of data, analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.
SOS: analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.
EJ: analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.
AJL: analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.
TTW: analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.
AD: conception and design of the study, analysis and interpretation of data, drafting the article, critical review, final approval of the submitted version.

Supplementary table 1 – Post hoc comparison between patients with punding and controls

	PD+pu (N = 21)	PD controls (N = 25)	Test statistic
HADS			
Total	18.2 (± 6)	13.1 (± 6)	**t(44)=-2.593; p=0.013**
Anxiety	10.4 (±3)	6.8 (± 4)	**t(44)=-2.932; p=0.005**
UPDRS part I	21.6 (± 9.9) N=20	13.2 (± 5) N=18	**t(36)=-3.168; p=0.003**
UPDRS part III	34.4 (± 12)	24.4 (± 14)	**t(44)=-2.503; p=0.016**
FAB	14.7 (± 3)	16.6 (± 2)	**U=147;p=0.008**

PD+pu – patients with Parkinson's disease and punding; PD-pu: patients with Parkinson's disease without punding; PD – Parkinson's disease; DBS – deep brain stimulation; DA – dopamine agonists; MAOI – monoamine oxidase inhibitors; LEDD – l-dopa equivalent daily dose; HADS – Hospital Anxiety and Depression Scale; UPDRS – Unified Parkinson's Disease Rating Scale; BIS11 – Barratt Impulsiveness Scale; AIMS - Abnormal Involuntary Movement Scale; FAB - Frontal Assessment Battery; MoCA - Montreal Cognitive Assessment; RBD – Rem Sleep Behaviour Disorder; AS – Apathy Scale. *Fisher's exact test. Significant results in bold.

Supplementary table 2 – Post hoc comparison between patients with punding and hobbyism

	Punding (N = 21)	Hobbyism (N = 26)	Test statistic
HADS			
Total	18.2 (± 6)	16 (± 5.8)	t(45)=-1.175; p=0.239
Anxiety	10.4 (± 3.9)	8.5 (± 3.7)	t(45)=-1.713; p=0.091
UPDRS part I	21.6 (± 9.9) N=20	17.7 (± 7.4) N=24	t(42)=-1.673; p=0.093
UPDRS part III	34.4 (± 12.9)	24.6 (± 8.1)	**t(45)=-3.011; p=0.003**
FAB	14.7 (± 3)	16.3 (± 1)	U=204; p=0.133

PD+pu – patients with Parkinson's disease and punding; PD-pu: patients with Parkinson's disease without punding; PD – Parkinson's disease; DBS – deep brain stimulation; DA – dopamine agonists; MAOI – monoamine oxidase inhibitors; LEDD – l-dopa equivalent daily dose; HADS – Hospital Anxiety and Depression Scale; UPDRS – Unified Parkinson's Disease Rating Scale; BIS11 – Barratt Impulsiveness Scale; AIMS - Abnormal Involuntary Movement Scale; FAB - Frontal Assessment Battery; MoCA - Montreal Cognitive Assessment; RBD – Rem Sleep Behaviour Disorder; AS – Apathy Scale. *Fisher's exact test. Significant results in bold.

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