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Abstract

Objectives: Impulse Control Disorders (ICDs) in Parkinson's disease (PD) have previously almost exclusively been considered to result from anti-parkinsonian medication. However, this biomedical perspective has failed to achieve a full understanding of the phenomenon and it is argued that a failure to consider psychological factors is a critical omission. Design: The present study examined the predictive relationship between ICDs in PD and a range of psychological measures, whilst controlling for a number of biomedical determinants. Main outcome measures: One hundred participants with idiopathic PD completed questionnaires that assessed demographic and clinical characteristics, psychological measures and the presence of ICDs (QUIP-RS). Results: Increased use of a 'negative' coping strategy, stronger illness identity, more emotional illness representations and stress were found to be significant predictors of ICDs, and different psychological predictors were associated with different ICDs. Medication was not found to predict ICDs in the presence of psychological factors, either when total treatment levels were considered or when agonist dose was considered alone. Conclusions: This study provides the first quantitative evidence of a predominant predictive relationship between psychological factors and ICDs in PD. The results suggest that psychological interventions may have a useful therapeutic role to play for ICDs in PD.

Key words: impulsivity; biomedical determinants; psychological determinants; coping; illness perceptions

Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by the loss of dopamine containing neurons primarily within the substantia nigra pars compacta of the midbrain (Obeso et al., 2000; Moore, 2003). The disease is typically characterised and diagnosed by a syndrome of bradykinesia, tremor, and muscular rigidity (Stern, 1988; Gelb, Oliver & Gilman, 1999). Research has tended to focus on the motor symptoms associated with PD, and although a number of non-motor symptoms are now recognised to be part of the disease, non-motor symptoms have received comparatively less attention (Chaudhuri, Healy, & Schapira, 2006; Gallagher, Lees, & Schrag, 2010). The most common non-motor symptoms are argued to include depression, anxiety, psychosis, sleep problems and impulse control disorders (ICDs; Chaudhuri et al., 2006; Chaudhuri & Schapira, 2009). Impulse control disorders have received growing attention as a feature of PD in recent years and include pathological gambling, compulsive buying, hypersexuality and compulsive eating (Giladi, Weitzman, Schreiber, Shabtai, & Peretz, 2007; Voon & Fox, 2007; Weintraub, 2008). Hobbyism (repeated ritualistic behaviours), punding (illogical ritualistic behaviours) and Dopamine Dysregulation Syndrome (personal escalation of medication doses) are also reported in the population (Ferrara & Stacy, 2008; Weintraub, 2008).

Aside from the range of difficulties in basic living skills people with PD often experience on a daily basis (Bramley & Eatough, 2005), ICDs carry their own consequences, frequently destabilising family life and causing significant financial losses (Voon & Fox, 2007). This added stressor multiplies the complexity of living with PD and has been found to have a significant detrimental effect on people's quality of life (Phu et al., 2014). Although ICDs are commonly under reported (Vilas, Pont-Sunyer, & Tolosa, 2011), it is estimated that between 10 and 14% of people with PD have ICDs (Giladi et al., 2007; Poletti et al., 2013; Weintraub,

Potenza, Siderowf, & Voon, 2010), compared to between 1 and 8% within the general population (Dell'Osso, Altamura, Allen, Marazziti, & Hollander, 2006). Where explicit comparisons have been made between people with PD and matched controls, ICDs are around twice as common in Parkinson's patients than in controls (Rodriguez-Violante, Gonzalez-Latapi, Cervantes-Arriaga, Camacho-Ordonez, & Weintraub, 2015), although the difference can be even greater (Perez-Lloret, Rey, Fabre, Ory, Spampinato, Brefel-Courbon, Montastruc, & Rascol, 2012).

To date, research into ICDs in PD has largely investigated associated risk factors from a biomedical perspective, concentrating on the role of dopamine replacement therapies, consisting of L-dopa and dopamine agonists used to treat motor symptoms (e.g. Voon, Potenza, & Thomsen, 2007). For example, research suggests that there is an increased prevalence of ICDs in people taking dopamine agonists, compared with those on other forms of dopamine replacement therapy (Weintraub, 2008; Weintraub et al., 2010), and ICDs have been shown to occur after an increase of dopamine agonist dosage over the course of the disease and to decrease after a reduction of dopamine agonist dosage in some (though not all) cases (Dodd et al., 2005). Within this theoretical framework, it is widely hypothesised that dopamine replacement therapies interact with the systems in the brain that reinforce behaviour and that engagement in impulsive-compulsive behaviours occurs as a result of the increased activation of these systems (Voon & Fox, 2007).

However, despite some support for dopamine agonist medication being linked to an increased prevalence of ICDs in PD (Lim, Evans, & Miyasaki, 2008), only a small percentage of those taking the medication are actually diagnosed with ICDs (Weintraub et al., 2006; 2010). The majority of patients do not develop ICDs even though they are often on very similar

dopamine replacement therapy treatment regimes to those who do (see for example Weintraub et al., 2010). Likewise, Tourette's syndrome, where a functional increase in dopamine-mediated transmission is considered a central component of the disorder (Minzer, Lee, Hong, & Singer, 2004), has not been associated with ICDs. Impulse control disorders themselves are not limited to PD and have been described in other chronic conditions such as angina and arthritis (Pietrzak, Morasco, Blanco, Grant, & Petry, 2007), dementia (Mendez, Bronstein, & Christine, 2000) and multiple sclerosis (Ceschin, Giannunzio, Favaro, & Santonastaso, 2010), where there is no explicit dopamine involvement. Finally, a solely biomedical perspective cannot easily provide insight into why a patient develops one ICD rather than another. So, although the case for the relationship between dopamine (and dopamine agonists) and ICDs in PD has some support, the field is still far from achieving a full understanding of the factors that underlie ICDs in PD.

The deficiencies in a solely biomedical perspective suggest that other important causative factors must be involved in the genesis of ICDs in PD. In relation to this, Lim et al. (2008) had a potentially critical insight when they speculated that how the patient copes with having a chronic illness may play a role in the development and maintenance of ICDs in PD. Qualitative work supports this: Hence, Delaney, Simpson and Leroi (2011) report that during interviews some PD patients with ICDs attribute the cause of their ICDs to the emotional impact of the disease and their way of coping with that impact. In these patients, ICDs reflect problem-focused coping strategies concerned with managing the problem (PD) that created stress in their lives, and emotion-focused strategies concerned with minimizing negative emotions associated with the stress through cognitive or emotional avoidance. A priori it seems plausible that a psychological perspective based on characteristics of the patient and the psychological consequences of living with a chronic, progressive illness might have much

to offer if one was seeking to understand the basis for the marked individual differences in the propensity for PD patients to develop ICDs in the presence of a common pharmaco-therapeutic background. Indeed, a person's perception of their illness is thought to have an important role to play in the adjustment a patient makes to a chronic condition (Leventhal, Nerenz, & Steele, 1984).

Given that the current biomedical approach to ICDs in PD has achieved an incomplete understanding of the phenomenon, whilst at the same time evidence suggests that psychological factors are likely to play an important in the genesis of ICDs in PD, there is an urgent need to more fully explore the relationship between psychological factors and ICDs in PD. Identifying the causal factors underlying ICDs in PD will have important implications for their treatment. Hence, in the current study, the relationships between coping, illness perceptions, anxiety, depression and stress in relation to ICDs were statistically examined using hierarchical block regression. In addition, studies to date have only looked at ICDs within the context of a clinical diagnosis and it is apparent that a significant proportion of PD patients do not report their difficulties with compulsive behaviour and therefore are not formally considered to have an ICD (e.g. Papay et al., 2011). Patients may also have compulsive behaviours that do not meet the full criteria for the relevant disorder in, for example, the DSM V (American Psychiatric Association, 2013). As a consequence, the current study was also concerned with exploring the predictors of subclinical ICDs in the PD population.

Method

Participants

Participants (N = 100) were recruited from across the United Kingdom (UK) following

ethical approval from the National Health Service (NHS) research ethics committee and local NHS Trust. Potential participants were eligible to take part in the study if they had a diagnosis of idiopathic PD from a neurologist (with or without a clinical ICD diagnosis), were able to provide informed consent and were English speaking. In line with policies for practice and research in the UK, capacity to take part in the research was assessed on an individual basis (Dobson, 2008; Department for Constitutional Affairs, 2005).

Participants were recruited through two possible routes, either from advertisements distributed by the charity Parkinson's UK (PDUK) or by letter of invitation from their neurologist at a regional specialist hospital in the North of England. One hundred and nineteen potential participants contacted us to take part (104 responding to the advertisements and 15 responding to letters sent to participants from their neurologist). Of those who contacted us to take part, one was ineligible and was excluded at screening level as they had not yet received a diagnosis of PD confirmed by a neurologist. One further participant was excluded from the analysis as they failed to complete some of the questionnaires. No other participants were excluded, although 17 failed to return their responses after contacting us to take part. Where there were missing values (this was rare), instructions specific to scales were followed. Of the final 100 participants who completed the questionnaires (PDUK, n = 88; neurologist, n = 12), 34 chose to complete them by post and 66 chose to complete them online. All participants reported a diagnosis of idiopathic PD from a consultant neurologist within the NHS.

Demographic and clinical predictor variables

Demographic information included: age; gender; marital status; occupational status; and years of education. Self-reported clinical information included: number of years since PD

diagnosis using a three point scale (in the last year, up to two years and over three years); years since symptom onset on a three point scale (during the last year, between two and three years and since diagnosis); if participants had a diagnosis of ICD (yes/no); and if they had ever had surgery to fit a deep brain stimulator for motor control problems (yes/no). Participants were also asked to provide information about their current PD medication and if they had noticed any changes since starting their current medication regime. Parkinson's disease medications were transformed into a Levodopa equivalent daily dose (LEDD) for each participant, using the equivalencies recommended by Tomlinson et al. (2010). As in Weintraub et al. (2010), analysis was conducted separately for total LEDD and for agonist dose converted to an equivalent LEDD.

Stage of illness and level of independence were assessed using self-report versions (see Schrag, Hovris, Morley, Quinn, & Jahanshahi, 2003; Schrag et al., 2007) of the Hoehn and Yahr (HY; Hoehn & Yahr, 1967) and Schwab and England (SE; Schwab & England, 1969) scales. The HY scale was developed to evaluate symptom severity for PD and its use is supported by the Movement Disorder Society Task Force for Rating Scales for Parkinson's disease (Goetz et al., 2004). The scale rates the stage of disease progression, ranging from 0 (no signs of disease) to 5 (wheelchair/bed bound). The SE scale was developed to measure the functional abilities of people with PD and the amount of perceived support that a person feels they require in order to complete daily tasks. Scores range from 0% (no control over swallowing, bladder or bowels – bed-ridden) to 100% (completely independent) with 10% increments. Although the scales are typically completed by clinicians, a self-administered version of each has been found to have strong correlations with clinician scores, with intra-class correlation coefficients reaching $r = .70$ for the HY scale and $r = .82$ for the SE scale (Schrag et al., 2003).

Demographic and clinical information are presented in Table 1. The majority of participants were married and retired. Most participants reported being educated to degree level or had post-statutory educational qualifications. An equal number of male and female participants took part in the study, with time since diagnosis typically over three years, and with PD symptom onset also generally under three years. The disability scale scores were consistent with a population in the earlier stages of the condition, with a reasonable level of independence. A range of medications for PD were reported. The majority of participants (70.0%) were taking Levodopa, although a number (70.0%) were taking dopamine agonists (most prevalent: ropinirole [39.0%], pramipexole [27.0%] and rotigotine [4.0%]). A proportion (39%) were taking an inhibitor, either of peripheral decarboxylase activity (3%; carbidopa), monoamine oxidase (MAO)-B activity (32.0%; most prevalent: rasagiline [25.0%] and selegiline [7.0%]) or catechol-O-methyl transferase activity (COMT; 6%; most prevalent entacapone [5.0%]). Most participants felt that they had noticed changes in their condition since onset or had experienced changes in their medication regime. Three participants reported having previously had deep brain stimulation (DBS) surgery for their condition. Eleven (11.0%) participants reported a current clinical ICD diagnosis.

Psychological predictor variables

Abbreviated Coping Orientations to Problems Experienced scale (Brief COPE). The Brief COPE (Carver, 1997) is an abbreviated version of the original COPE inventory (Carver, Scheier, & Weintraub, 1989), consisting of 28 statements divided into 14 subscales: active coping; planning; positive reframing; acceptance; humour; religion; use of emotional support; instrumental support; self-distraction; denial; venting; substance use; behavioural disengagement; and self-blame. The subscales have a high level of consistency and reliability ranging from $\alpha = .50$ for the venting subscale to $\alpha = .90$ for substance misuse (Carver, 1997).

The Brief COPE has been used in studies investigating PD and found to measure coping within acceptable limits (Dobkin et al., 2011; Simpson, Lekwuwa, & Crawford, 2013).

Depression, Anxiety and Stress Scale (DASS). The DASS (Lovibond & Lovibond, 1995) is comprised of three self-report scales to measure current levels of depression, anxiety and stress. As the DASS subscales can be used independently, only the stress scale was used in the present study. The scale consists of 14 statements which are rated using a four-point Likert scale, from 0 ("Did not apply to me at all") to 3 ("Applied to me very much, or most of the time") based on experiences over the past week. The stress subscale score is calculated by summing the items together with a maximum score of 42. Scores below 14 are considered to be levels that most people would experience. The stress subscale has been found to have high internal consistency ($\alpha = .93$; Crawford & Henry, 2010) and has been used with people with PD in previous research (Simpson, Haines, Lekwuwa, Wardle, & Crawford, 2006). Although the depression and anxiety scales on the DASS have been used in PD research, a number of the items have been argued to be confounded by physical symptoms of PD (Marinus, Leentjens, Visser, Stiggelbout, & van Hilten, 2002). Therefore the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) was considered more appropriate to measure anxiety and depression in this population.

Hospital Anxiety and Depression Scale (HADS). The HADS (Snaith, 2003; Zigmond & Snaith, 1983) is 14-item self-report measure comprised of two subscales for current levels of anxiety and depression, scored on four-point Likert scales from 0 to 3, with higher scores representing greater levels of anxiety or depression. Each scale has a maximum score of 21, with scores between eight and ten indicating raised levels of anxiety or depression and scores above 10 indicating 'probable caseness' for anxiety or depression. The anxiety and

depression scales have been found to demonstrate high internal consistency in 15 reviewed studies (Cronbach alphas from α = .68 to α = .93 for anxiety and α = .67 for depression (Bjelland, Dahl, Haug, & Neckelmann, 2002). Similar results have been found for test-retest reliability in a population with PD (Marinus et al., 2002).

Life History of Impulsive Behaviour questionnaire (LHIB-Q20). The LHIB-Q20 (Coccaro & Schmidt-Kaplan, 2012) is a self-report version of the Life History of Impulsive Behaviours interview (Schmidt, Fallon, & Coccaro, 2004). The scale consists of 20 statements relating to adult life rated on a six-point Likert scale from 0 ("never") to 5 ("happened too many times to remember"). There are currently no normative data for the scale, however a higher score indicates a more impulsive life history. High scores on the self-report measure have been found to be consistent with diagnoses of ICDs made by clinicians using diagnostic criteria (Coccaro & Schmidt-Kaplan, 2012). The authors report high internal reliability for the LHIB-Q20 (α = .95).

Illness Perceptions Questionnaire – Revised (IPQ-R). The IPQ-R (Moss-Morris et al., 2002) is used to assess individuals' beliefs and understandings about their illnesses. Validity and reliability have been confirmed across a range of clinical populations (see Llewellyn, McGurk, & Weinman, 2007). The IPQ-R includes subscales for: illness identity; cause of illness; timeline (chronic and cyclical); consequences; control (personal and treatment); coherence and emotional representations. With the exception of illness identity, all items are scored on a five-point Likert scale from 1 ("strongly disagree") to 5 ("strongly agree"). The Identity subscale consists of illness symptoms, awarded a score of one if the symptom is present and related to the illness or zero if not. Higher scores on each subscale represent stronger beliefs for the domain. Good internal consistency for each of the subscales are

reported, ranging from α = .79 for the cyclical timeline to α = .89 for the chronic timeline (Moss-Morris et al., 2002). The authors state that adaptations should be made to the Identity and Cause subscales to describe illness specific symptoms and causes. In the present report, the Identity and Cause subscales were modified, based on and extending the changes made to the subscales in a previous study using a PD population, where 13 items were added to the Cause subscale and 18 were added to the Identity subscale (see Simpson et al., 2013a; Figures S1 and S2 in the supplemental online material).

Outcome variable

Questionnaire for Impulsive-comp*ulsive disorders in Parkinson's* disease – Rating Scale (QUIP-RS). The QUIP-RS (Weintraub et al., 2012) is a screening tool designed to measure the severity of ICD symptoms and assist with ICD diagnosis. Four subscales for ICDs (gambling, sexual compulsions, compulsive buying and compulsive eating) have possible scores between 0 and 16 and suggested cut-offs (scores validated as indicative of ICDs via clinical interview) as follows: gambling (≥6); compulsive buying (≥8); sexual compulsions (≥8); compulsive eating (≥7); and combined ICDs (≥10). The QUIP-RS also includes measures for hobbyism (involvement in organised activities like writing or computer use) and punding (repeating activities like cleaning). The two were combined into one subscale (hobbyism-punding; range = 0-32; cut-off ≥7) due to the significant overlap reported by the authors (Weintraub et al., 2012). Good inter-rater (α = .93) and retest (α = .87) reliabilities are reported for the questionnaire (Weintraub et al., 2012). Within the current study, the four ICD subscales and hobbyism-punding subscale were used as outcome variables in order to understand the relationship between the predictor variables and each type of ICD. The combined ICD score was used to establish the overall state impulsive characteristics of the sample and determine the number of participants reporting ICDs above the clinical cut-off.

Procedure

Data collection. Participants initially contacted the research team in response to advertisements distributed by PDUK or by letter of invitation from their neurologist. Following initial contact, they were screened for eligibility over the telephone by a researcher (JKG) and those with a diagnosis of idiopathic PD were provided with the option to complete questionnaires online or by post. Those choosing to complete the questionnaires by post were sent the questionnaire pack, consent form and pre-paid envelope. Those choosing to complete the questionnaires online received an email with a link to the questionnaires and consent form (hosted by SurveyGizmo). Participants were informed that the questionnaire pack would take about 45 minutes to complete, that it could be completed in sections over a maximum of one week and that they could withdraw from the study at any time without affecting their medical care. No participants reported difficulties completing the questionnaires and all questionnaires were completed within the week time frame.

Statistical Analysis

Statistical analyses were carried out using IBM SPSS Statistics (Version 21 for Macintosh). Comparisons between participants with higher and lower scores on the QUIP-RS were carried out using independent samples t-tests and hierarchical block regression analyses were conducted to investigate the relationship between predictor variables and the ICD outcome measures. Although for regression analysis, only dependent variables need to be normally distributed (Field, 2009), the QUIP-RS ICD subscale scores were not normally distributed (Z skewness > 1.96), therefore transformation was required (Tabachnick & Fidell, 2007). Optimal transformations were carried out for the dependent variables using the 'Box-Cox' procedure, which maximises the normality achievable via transformation of the data (Osborne, 2010). Spearman's correlations were undertaken where variables did not meet

assumptions of normal distribution to investigate the relationship between all demographic, clinical, psychological, coping and illness perceptions predictor variables and the ICD outcome variables (see Tables 2 and 3 for correlation coefficients). Only predictor variables significantly correlated with the ICD outcome variables at $p < .01$ were entered into the separate regression models due to the high number of correlations between variables (Edgar & Skinner, 2003; Simpson et al., 2013a; Simpson, Lekwuwa, & Crawford, 2014). Separate regression analyses were completed for each ICD (gambling, sexual compulsions, compulsive buying, compulsive eating and hobbyism-punding behaviours). Given the low percentage of people with PD experiencing two or more ICDs (3.9%; Weintraub et al., 2010), the relationship with the combined ICD score was not examined. Where predictor variables correlated at the required level with the outcome variables, blocks were conceptualised as follows:

Block 1: Demographic variables (gender, age, years of education, relationship status, employment status)

Block 2: Clinical variables (stage of illness, level of independence, years since diagnosis, years since onset, LEDD total, LEDD agonist, DBS, changes due to medication)

Block 3: Psychological variables (coping, trait impulsivity)

Block 4: Wellbeing (anxiety, depression, stress)

Block 5: Illness beliefs (IPQ-R)

Prior to interpretation of the regression analyses, variables were checked for assumptions required for multiple regression analysis (i.e. multicollinearity, collinearity, homoscedasticity and independent errors; see Tabachnick & Fidell, 2007). Correlations between predictors did not indicate high levels of multicollinearity in the sample. Where there was evidence of correlation between predictor variables, moderation analysis was carried out to further

investigate the interaction, centring the variables where necessary. Residual scatter plots were drawn and did not indicate any violations for normality. Additionally, linearity and homoscedasticity assumptions were met. No evidence of auto-correlation using the Durbin-Watson statistic (Durbin & Watson, 1971) in any of the models reported above was observed. Casewise diagnostics did not indicate any significantly influential cases, within what would be expected for general populations (Field, 2009).

Results

Participant characteristics

Means and standard deviations for psychological and ICD variables are presented in Table S1 in the supplemental online material. Scores on HADS revealed 39.0% of the sample to have raised levels of anxiety (with 19.0% in the 'probable caseness' range) and 32.0% of the sample to have raised levels of depression (with 7.0% in the 'probable caseness' range). On the DASS stress subscale, three participants (3.0%) were within the 'mild' range, with all remaining participants in the 'normal' range. Variation was found in the extent to which various coping strategies were endorsed, the most frequently used strategies being acceptance, active coping, planning, and emotional and instrumental support. Responses to questions concerning illness beliefs showed stronger beliefs about health and psychological attributions for the cause of PD. Personal control of PD appeared to be more important than treatment control and participants held strong beliefs about the consequences of their illness. More emotional representations about the illness were reported alongside beliefs that the illness would last a long time.

Despite the slightly lower than expected presentation of confirmed clinical ICD diagnoses (11.0%), responses for the combined ICD subscales (total QUIP-RS scores) showed a high

proportion of people in the 'clinical' range (combined score ≥10; n = 59, 59.0%). A number of impulsive behaviours in the clinical range were described using individual ICD scales within the sample. There were reports of hobbyism-punding (n = 45, 45%), compulsive eating (n = 29, 29.0%), sexual compulsions (n = 16, 16.0%) and compulsive buying (n = 11, 11.0%), indicating the presence of a range of compulsive behaviours in the sample. Gambling (n = 3, 3.0%) was the least reported ICD.

Considering those participants with total QUIP-RS scores in the clinical range versus those with scores below the clinical range, participants in the higher combined ICD group reported significantly greater scores on measures of emotional distress (anxiety, depression and stress) and increased use of more problematic coping strategies (self-distraction, substance use and self-blame; Table 2). They also reported more dominant emotional illness representations, increased perceived number of symptoms related to PD and beliefs that their illness was related to their environment. There were no significant differences between the total LEDD in those with higher and lower total QUIP-RS scores (mean ± 1 SD: 804.85 mg ± 501.87 vs 629.16 ± 480.17 mg respectively; $t[98] = 1.752$, $p > .05$, $d = 0.354$), or between the agonist equivalent LEDD in those with higher and lower total QUIP-RS scores (618.81 mg ± 593.15 vs 522.36 mg ± 512.35 respectively; $t[98] = .845$, $p > .05$, $d = .171$). Furthermore, the frequency of ICDs was not significantly different in those taking dopamine agonists and those not taking dopamine agonists ($\chi^2 = .096$, $p > .05$, odds ratio = 1.147). When different combinations of drug treatment (Levodopa only; Levodopa + agonist; Levodopa + inhibitor(s); Levodopa + agonist + inhibitor(s); agonist + inhibitor; agonist only and inhibitor only) were compared to each other and participants receiving no medication using one-way ANOVA, there was a significant main effect of treatment ($F(7,92) = 2.428$, $p < .05$, $\eta^2 = .156$). Although there was a trend for participants in the no medication group to have the

lowest QUIP-RS scores (mean ± 1 SD, 4.00 ± 3.23) and those in the Levodopa only group to have the highest (mean ± 1 SD, 16.20 ± 7.61), none of the post hoc comparisons (using Tukey's Honest Significant Difference test) were significant.

Multiple Regression Analyses

Correlations were carried out between each of the predictor variables and outcomes as described above (Tables 3 and 4). Multiple regression analyses were then carried out for the individual ICD subscales to further understand the relationships with the demographic, clinical and psychological predictors. Table 5 provides beta weights and standard error scores for each of the models below.

Hobbyism-punding. No clinical variables significantly correlated at the specified level with the hobbyism-punding outcome variable and these were therefore excluded from the model. Gender (Table 3), substance use and self-distraction coping, and illness cognitions for cause (Table 4) were correlated at the specified level with hobbyism-punding, hence gender was entered in block 1 and accounted for 6.4% of variance ($R2 = .064$, $p = .011$). Substance use and self-distraction coping were entered into block 2, accounting for 12.3% additional variance ($R2 = .186$, $R2$ change $= .123$, $p = .001$). Illness cognitions for cause (chance and psychological attributions) were entered in block 3 and accounted for 4.1% additional variance ($R2 = .228$, $R2$ change $= .041$, $p = .086$). Regression coefficients for predictors in the full model showed male gender ($\beta = .221$, $t = 2.385$, $p = .019$), increased substance use coping ($\beta = .270$, $t = 2.918$, $p = .004$) and increased self-distraction coping ($\beta = .205$, $t = 2.219$, $p = .029$) to significantly predict hobbyism-punding scores. The total variance explained by this model was 18.6% ($F(3,96) = 7.319$, $p < .001$; Table 5).

Compulsive eating. No significant relationships at the specified level were found for clinical or demographic variables with the compulsive eating outcome variable (Table 3), however substance use coping and stress were correlated to the required level (Table 4). Substance use coping was therefore entered as block 1, accounting for 8.1% variance (R2 = .081, p = .004). Stress was then entered in block 2 and accounted for 5.2 % additional variance (R2 = .132, R2 change = .052, p = .018). Regression coefficients for predictors in the full model revealed increased substance use coping (β = .219, t = 2.226, p = .028) and increased level of stress (β = .237, t = 2.411, p = .018) to be significant predictors of eating compulsions with the model explaining 13.2% of the total variance (F(2,97) = 7.407, p = .001; Table 5).

Sexual compulsions. Significant relationships at the specified level were found for total LEDD, level of independence and gender with the sexual compulsions outcome variable (Table 3), and these were entered into block 1, explaining 21.5% of variance (R2 = .215, p < .001). Only gender was a significant independent predictor (R2 = .060, p < .05). Self-distraction coping was entered into block 2 and accounted for 6.5% additional variance (R2 = .280, R2 change = .065, p = .004). Illness cognitions (identity, emotional representations and consequences) were entered in block 3, accounting for 11.14% additional variance (R2 = .392, R2 change = .111, p = .001). Regression coefficients for predictors in the full model showed male gender (β = .305, t = 3.474, p = .001), stronger illness identity (β = .258, t = 2.654, p = .009) and more emotional illness representations (β = .315, t = 3.331, p = .001) to be significant predictors of sexual compulsions in the sample, whilst level of independence, total LEDD, self-distraction coping and consequences were no longer significant predictors. The total variance explained by the model was 39.2% (F(7,92) = 8.460, p < .001; Table 5).

Given the interest in the literature concerning the relationship between ICDs in PD and

medication status (see Introduction), and the significant relationship at the specified level for total LEDD with the sexual compulsions outcome variable, the possibility was explored that total LEDD may moderate the effects of certain predictors on sexual compulsions. Levodopa Equivalent Daily Dose correlated at a low level (< .2) with most psychological predictors. However, correlations were significant with illness identity ($r = .276$, $p < .01$) and consequences ($r = .233$, $p < .05$), so interaction terms for these with LEDD were entered as additional predictors in the final model. Although these interaction terms accounted for 1.2% of additional variance, this was not significant ($R2 = .404$, $R2$ change $= .012$, $p = .384$), and neither emerged as significant independent predictors.

Compulsive buying. No significant relationships were found at the specified level for clinical or demographic variables with the compulsive buying outcome variable (Table 3), although substance use coping and stress were correlated to the required level (Table 4). Substance use coping was therefore entered in block 1 and accounted for 10.3% variance ($R2 = .103$, $p = .001$). Stress was then entered in block 2 and accounted for 3.2 % additional variance ($R2 = .135$, $R2$ change $= .032$, $p = .061$). Regression coefficients for predictors in the full model revealed increased substance use coping was the sole significant predictor ($\beta = .321$, $t = 3.353$, $p = .001$) of buying compulsions. The total variance explained by the model was 10.3% ($F(1,98) = 11.241$, $p = .001$; Table 5).

Gambling. As discussed earlier, only a small number of participants (n = 3) reported QUIP-RS scores in the clinical range for the gambling subscale and consequently no significant relationships at the specified level were found with any of the predictor variables.

Discussion

Impulse Control Disorders in PD have largely been considered from a biomedical perspective up to now, with PD medication seen as the primary causative factor. However, evidence suggests that psychological factors may also play an important role in the genesis of these disorders – a contention which receives considerable support from the current study. Illness beliefs in the form of a stronger illness identity (identification with the illness label), more psychological distress (increased emotional representations and stress) and 'negative' coping styles (more substance abuse coping and self-distraction coping) were found to explain a significant amount of variance in ICDs. The overall findings of the current study provide the first quantitative evidence for relationships between coping, illness beliefs, psychological distress and ICDs in people with PD. Previous research has hinted at the importance of psychological factors in ICDs in PD. For example, Voon and colleagues (2007; 2011) suggest that certain psychological factors such as strong novelty seeking traits and lack of concern for the future are associated with ICDs in PD. However, the current findings are the first demonstration that psychological factors can be the predominant predictors of ICDs in PD.

Although previous research has suggested ICDs to be present in 13.0-14.0% of people with PD when assessed by clinical interview (Weintraub et al., 2010; Papay et al., 2011), the current sample reported a significantly higher level, reaching 59.0% on a self-report measure**,** the QUIP-RS, when using clinically validated cut offs for ICDs. Similarly high (>30%) prevalence rates have been reported in other studies using the QUIP-RS (Papay et al., 2011; Lim et al., 2011; Goerlich-Dobre et al., 2014). Given that the diagnosed prevalence of ICDs in the current study was close to that reported elsewhere (11.1%), the greater level of self-reported ICDs supports the theory that ICDs are under reported and often may not always be recognised by clinicians (Vilas et al., 2011; Voon & Fox, 2007).

Perhaps the most significant point of departure of the current results from previous findings is in relation to dopamine replacement therapies as predictors of ICDs in PD. Previous reports suggest that there is an increased prevalence of ICDs in people taking dopamine agonists, compared with those on other forms of dopamine replacement therapy (e.g. Weintraub, 2008; Weintraub et al., 2010; Hurt et al., 2014), and ICDs have been shown to occur after an increase of dopamine agonist dosage over the course of the disease and to decrease after a reduction of dopamine agonist dosage in some cases (Dodd et al., 2005). However, in the present study, dopamine replacement therapies (total LEDD and dopamine agonist dose converted to a LEDD) were not associated with ICDs. Participants with higher total QUIP-RS scores did not have significantly different total LEDD levels or dopamine agonist dose levels to those with lower scores, nor were they more likely to be taking agonist medication. While LEDD was significantly correlated at the specified level with sexual compulsions, LEDD was not a significant independent predictor (when entered alongside level of independence and gender). Although there are many previous reports which link ICDs in PD to dopamine replacement therapies (especially dopamine agonists), not all previous studies concur. In PD patients prescribed several forms of dopamine replacement therapy in conjunction, overall LEDD has sometimes not been found to predict ICD development (Voon et al., 2007; Isaias et al., 2008). Furthermore, although some studies have reported that dopamine agonists create an increased risk of developing ICDs others have not demonstrated this relationship (Lee et al., 2010; Hurt et al., 2014). Delaney, Leroi, Simpson and Overton (2012) hypothesise that medication may be a 'susceptibility multiplier' which operates in conjunction with psychosocial factors to determine whether the psychological distress (depression, anxiety and stress) caused by living with a chronic illness exceeds some threshold and leads to the development ICDs. The results of the present study suggest that psychological factors alone may be sufficient to exceed that threshold.

The present results also suggest that particular constellations of psychological factors may determine which ICDs will emerge. Where relevant, demographic and clinical variables were entered into the regressions early on and accounted for a proportion of the variance (generally $R2 < .2$) for hobbyism-punding and sexual compulsions. However, adding in psychological variables increased the amount of variance accounted for by the model in both cases. Although self-distraction, substance use, stress and illness identity had shared influences on hobbyism-punding and sexual compulsions, the latter was also associated with illness consequences and emotional representations. Substance use alone was associated with compulsive buying and when twinned with stress, substance abuse was associated with compulsive eating.

Clinical implications: At present, the primary clinical response to ICDs in PD is to reduce the dopamine agonist medication (Macphee & Carson, 2013). Motor symptoms are sometimes significantly worsened when dopamine agonist medication is reduced, even though L-dopa doses may be increased to compensate (Lim et al., 2008). Therefore, people who develop ICDs in PD may be reluctant to discontinue dopamine agonist use, due to the motor symptom benefits. Furthermore, manipulating dopamine agonist therapy is not always successful in treating ICDs (Dodd et al., 2005). The finding that psychological factors have a significant predictive role in ICDs in PD suggests that there may be a role for other therapeutic approaches, such as psychological interventions, to be used alongside pharmacotherapies, and initial indications are that these approaches – such as Cognitive Behaviour Therapy (CBT) (Jiménez-Murcia et al., 2012; Okai et al., 2013) and support groups (Kurlan, 2004; Mamikonyan et al., 2008) - can be effective. However, our understanding of psychological involvement in ICDs in PD is still in its infancy, although the present study suggests that the current approach to using CBT in ICDs in PD (where ‘second wave’ therapies based on

challenging dysfunctional thoughts have been used – therapies that in their manual-based form have a major focus on symptom reduction, e.g. Cukor, 2007) may not be appropriate. Our study suggests that interventions need to take a different tack and explore the purpose behind the expressed ICDs (e.g. coping….). These behaviours are clearly far from ideal, however they fulfil an adaptive role to play for the people involved. A number of third wave cognitive behavioural therapies may be more appropriate for managing the psychological distress and problematic coping patterns associated with PD. Mindfulness based interventions and Acceptance and Commitment Therapy (ACT) have begun to receive attention within chronic illness research (e.g. Bohlmeijer, Prenger, Taal, & Cuijpers, 2010; Dahl, Wilson, & Nilsson, 2004; Fitzpatrick, Simpson, & Smith, 2010). Interventions have focused on adjustment to illnesses such as diabetes, drug refractory epilepsy and chronic pain (Gregg, Callaghan, Hayes, & Glenn-Lawson, 2007; Lundgren, Dahl, Melin, & Kies, 2006; Wetherell et al., 2011). The focus of intervention involves working with adjustment issues related to acceptance of the chronic illness and focusing on living in the present moment rather than focusing on the illness symptoms. Results have shown significant reductions in psychological distress as well as improvements in more adaptive coping strategies. There remains a demand for further research within PD to adapt appropriate therapies to help people develop more helpful beliefs about their illness and develop more positive coping strategies. Given the demonstrated importance of psychological factors in ICDs in PD, psychological therapies in the future could form the basis for a significant improvement in the management of these problematic symptoms. Indeed, further use of support groups may allow the opportunity to for patients to discuss and explore the personal story behind their behaviour without a priori medicalisation, and at the same time reduce psycho-emotional problems arising from stigma (Simpson, McMillan, & Reeve, 2013; Simpson, & Thomas, 2015) that may be themselves be disabling via depression (Simpson, et al., 2013b) and hence the subsequent impact on quality

of life (Simpson et al., 2014).

Limitations: While the current report represents a useful addition to understanding the determinants of ICDs in PD, there are limitations worth noting. Firstly, on the whole the correlation coefficients and R2 values in the current study, although often significant, were in many cases reasonably small (r was often between .2 and .3, and R2 often < .2). This suggests that there are ultimately a large number of factors associated with ICDs in PD, alongside those reported here. Previous work by other authors has identified predictive relationships between personality factors, gambling problems in first-degree relatives and current smoking status, and ICDs in PD (Weintraub et al., 2010; Voon et al., 2011). We hypothesise that this list can be augmented by other factors, including additional psychological factors such as social support. In short, the relatively small correlation coefficients and R2 values point to the interesting conclusion that ICDs in PD have a multi-factorial cause.

Secondly, in the multiple regression analyses, we pre-screened all predictors for significant correlations with the DV and entered only those with significant (< .01) univariate p values. Although this data driven method of subset selection can be defended for an exploratory study like the present one, other methods are also possible, although their requirement for a greater a priori understanding of the relationship between predictors and the outcome measure, and/or a larger sample size, confine these approaches to the future. Hence, Steyerberg, Eijkemans, Harrell, and Habbema (2001) conclude that predictors are best selected on the basis of 'external information', which is lacking for psychological predictors of ICDs in PD. Approaches like stepwise regression would impose the least bias, however with our large number of predictors and 100 participants, we would greatly exceed the

maximum number of predictors to be statistically robust (Harris, 1985 - the number of participants should exceed the number of predictors by at least 50; Green, 1991 - N > 50 + 8m, where m is the number of predictors).

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Table 1
Participant demographic and clinical information (N = 100)

Variable	M (SD) or n (%)
Age	64.39 (9.73)
Gender (male)	50 (50%)
Years of education	14.22 (2.29)
Marital status	
Single	1 (1%)
Married	79 (79%)
Living with partner	6 (6%)
Divorced/separated	8 (8%)
Widowed	6 (6%)
Employment status	
Employed (Full-time/Part-time)	12/11 (12%/11%)
Self-employed	3 (3%)
Student	1 (1%)
Retired	69 (69%)
Unemployed	4 (4%)
DBS surgery for PD	3 (3%)
Confirmed clinical ICD diagnosis	11 (11%)
Years since PD diagnosis	
< 1 year	3 (3%)
1-3 years	23 (23%)
> 3 years	74 (74%)
Years since symptom onset	
< 1 year	12 (12%)
1-3 years	50 (50%)
> 3 years	38 (38%)
Hohen and Yahr	1.78 (1.32)
Schwab and England	76.40 (17.34)
Levodopa equivalent daily dose [LEDD] (mg)	732.83 (498.26)
Dopamine agonist daily dose [as LEDD] (mg)	579.26 (560.76)
Changes due to medication	86 (86%)

Note. DBS = Deep brain stimulation surgery.

Table 2
Between group analysis for higher (n = 59) and lower (n = 41) combined Impulse Control Disorder responses

Variable	High M (SD)	Low M (SD)	t	p	d
COPE – Self-distraction	4.78 (1.63)	3.73 (1.64)	3.15**	.002	.636
COPE – Substance use	2.73 (1.22)	2.12 (0.46)	3.05**	.003	.616
COPE – Self-blame	3.42 (1.74)	2.59 (1.07)	2.74**	.007	.554
HADS – Anxiety	7.51 (4.10)	5.66 (3.37)	2.38*	.019	.481
HADS – Depression	6.64 (3.53)	4.49 (2.84)	3.25**	.002	.657
DASS – Stress	11.42 (7.61)	7.27 (5.29)	3.02**	.003	.610
IPQ-R – Identity	16.66 (6.23)	12.83 (7.22)	2.83**	.006	.572
IPQ-R – Consequences	24.21 (3.79)	22.34 (5.54)	2.01*	.047	.406
IPQ-R – Emotional representations	19.85 (4.43)	17.61 (4.92)	2.37*	.020	.479
IPQ-R – Environmental attributions	11.14 (3.36)	9.64 (3.11)	2.25*	.026	.455

Note. COPE = Abbreviated coping orientations to problems experienced scale. HADS = Hospital anxiety and depression scale. DASS = Depression, anxiety and stress scale. IPQ-R = Illness perceptions questionnaire revised. d = Cohen's d. *p < .05; **p < .01

Table 3
Correlation coefficients for demographic, clinical predictors and Impulse Control Disorder variables (N = 100)

Variable	Gamb.	Sex	Buying	Eating	HBY-P
Age	0.067	-0.017	-0.102	0.014	0.088
Gender	0.027	**.405*** **	-0.007	0.037	**.286** **
Years of education	0.049	-0.061	-0.07	-0.128	-0.131
Relationship status [a]	0.012	0.169	-0.134	-0.111	0.043
Employment status [a]	-0.149	-0.014	0.077	-0.107	-0.051
DBS for PD [a]	0.065	0.107	0.134	-0.059	0.001
Years since diagnosis [b]	-0.094	-0.063	-0.037	-0.052	-0.126
	0.035	-0.036	0.011	0.028	-0.049
	0.003	0.059	0.004	-0.007	0.096
Years since symptom onset [b]	-0.131	-0.042	-0.144	-0.018	-0.165
	0.045	-0.044	.211*	0.087	0.066
	0.042	0.074	-0.121	-0.078	0.043
Hoehn and Yahr	0.033	-0.064	-0.011	-0.043	-0.003
Schwab and England	-0.134	**-.269** **	0.086	0.023	-0.137
Levodopa equivalent daily dose	0.089	**.264** **	0.072	0.032	0.113
Dopamine agonist daily dose	0.013	0.161	-0.063	0.097	0.091
Medication changes	0.034	0.167	0.058	0.123	0.047

Note. DBS = deep brain stimulation surgery. a = dummy coded (0 = not in a relationship/not working/not DBS, 1 = in a relationship/working/DBS). b = dummy coded (<1yr, 1-3yrs or >3yrs). Gamb. = Gambling. HBY-P = Hobbyism-punding. *p < .05; **p < .0; ***p < .001

Table 4
Correlation coefficients for psychosocial predictor and Impulse Control Disorder variables (N = 100)

Predictor variable	Gamb.	Sex	Buying	Eating	HBY-P
COPE – Active coping	-0.002	0.038	-0.012	0.068	-0.132
COPE – Planning	0.048	-0.021	0.024	0.114	-0.063
COPE – Positive reframing	-0.042	-0.068	-0.103	0.012	-0.087
COPE – Acceptance	-0.17	-0.073	-0.1	-0.027	-0.127
COPE – Humor	-0.069	-0.082	-0.099	-0.018	-0.088
COPE – Religion	-0.083	0.008	-0.061	0.1	-0.015
COPE – Using emotional support	0.022	0.003	-0.193	-0.094	-0.08
COPE – Using instrumental support	-0.007	0.14	-0.099	-0.074	-0.088
COPE – Self-distraction	0.182	**.293****	0.079	.242*	**.264****
COPE – Denial	0.054	0.131	0.042	0.076	0.083
COPE – Venting	0.138	0.075	0.112	0.097	0.096
COPE – Substance use	.197*	.252*	**.318****	**.283****	**.285****
COPE – Behavioral disengagement	0.091	0.194	0.059	0.063	0.169
COPE – Self-blame	-0.04	0.184	0.171	0.192	0.018
LHIB-Q20	0.185	.209*	0.145	0.15	.204*
HADS – Anxiety	0.141	.246*	0.113	0.176	.216*
HADS – Depression	0.063	.234*	0.119	.241*	0.119
DASS – Stress	0.057	.215*	**.273****	**.293****	.233*
IPQ-R – Identity	0.173	**.375****	0.143	0.134	.232*
IPQ-R – Timeline (acute/chronic)	-0.003	0.051	-0.036	0.035	-0.071
IPQ-R – Timeline (cyclical)	0.124	.228*	0.186	0.148	.242*
IPQ-R – Personal control	0.042	0.022	0.139	0.09	-0.1
IPQ-R – Treatment control	-0.142	-0.148	0.014	-0.04	-0.161
IPQ-R – Coherence	-0.098	0.046	-0.073	-0.089	-0.173
IPQ-R – Consequences	0.133	**.271****	0.027	0.136	0.061
IPQ-R – Emotional representations	0.1	**.358*****	0.146	.198*	0.139
IPQ-R – Psychosocial attributions	0.061	0.174	0.098	0.112	**.272****
IPQ-R – Health attributions	0.047	0.048	0.127	0.127	0.087
IPQ-R – Behavioral attributions	0.135	0.074	0.076	0.052	0.175
IPQ-R – Environmental attributions	-0.021	0.111	0.196	0.116	0.128
IPQ-R – Chance attributions	0	.222*	0.065	0.073	**.273****

Note. COPE = Abbreviated coping orientations to problems experienced scale. LHIB-Q20 = Life history of impulsive behavior questionnaire. HADS = Hospital anxiety and depression scale. DASS = Depression, anxiety and stress scale. IPQ-R = Illness perceptions questionnaire revised. Gamb. = Gambling. HBY-P = Hobbyism-punding. *p < .05; **p < .01; ***p < .001

Table 5
Summary of multiple regression analyses predicting Impulse Control Disorders (N = 100)

	Compulsive eating			Hobbyism-punding		
Predictors	B	SE (B)	*β*	B	SE (B)	*β*
BLOCK 1						
Gender				.526	.204	.252*
COPE – Substance use	.160	.055	.284**			
R^2	.081**			.064*		
BLOCK 2						
Gender				.460	.193	.221*
COPE – Self-distraction				.126	.057	.205*
COPE – Substance use	.124	.056	.219*	.277	.095	.270**
DASS - Stress	.019	.008	.237*			
R^2	.132*			.186**		
BLOCK 3						
Gender				.449	.192	.215*
COPE – Self-distraction				.093	.060	.151
COPE – Substance use				.236	.097	.230*
IPQ-R – Cause (Psychological)				.011	.017	.066
IPQ-R – Cause (Chance)				.069	.034	.191*
R^2				.228**		
FINAL MODEL						
Gender				.460	.193	.221*
COPE – Self-distraction				.126	.057	.205*
COPE – Substance use	.124	.056	.219*	.277	.095	.270**
DASS – Stress	.019	.008	.237*			
IPQ-R – Identity						
R^2	.132*			.186**		

	Sexual compulsions			Compulsive buying		
Predictors	B	SE (B)	*β*	B	SE (B)	*β*
BLOCK 1						
Gender	.573	.167	.329**			
SE	-.009	.005	-.181			
LEDD	< .001	< .001	.128			
COPE - Substance use				.031	.009	.321**
R^2	.215**			.103**		
BLOCK 2						
Gender	.550	.161	.315**			
SE	-.008	.005	-.165			
LEDD	< .001	< .001	.120			
COPE – Self-distraction	.132	.045	.257			
COPE - Substance use				.026	.009	.270**
DASS - Stress				.003	.001	.186
R^2	.280**			.135		
BLOCK 3						
Gender	.532	.153	.305**			
SE	-.005	.004	-.099			
LEDD	< .001	< .001	.107			
COPE – Self-distraction	.088	.047	.171			
IPQ-R - Identity	.033	.012	.258**			
IPQ-R - Emotional representations	.058	.017	.315**			
IPQ-R - Consequences	-.035	.021	-.183			
R^2	.392**					
FINAL MODEL						
Gender	.532	.153	.305**			
SE	-.005	.004	-.099			
LEDD	< .001	< .001	.107			

COPE – Self-distraction	.088	.047	.171			
COPE – Substance use				.031	.009	.321**
IPQ-R – Identity	.033	.012	.258**			
IPQ-R – Emotional representations	.058	.017	.315**			
IPQ-R – Consequences	-.035	.021	-.183			
R^2	.392**			.103**		

Note. LEDD = Levodopa equivalent daily dose. COPE = Abbreviated coping orientations to problems experienced scale. HADS = Hospital anxiety and depression scale. DASS = Depression, anxiety and stress scale. IPQ-R = Illness perceptions questionnaire revised. QUIP-RS = Questionnaire for impulsive-compulsive disorders in Parkinson's disease. *p < .05; **p < .01