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Prevalence and Treatment of Impulse Control Behaviours in Parkinson's Disease: A Meta- Analysis and Systematic Review

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Abstract

Objective

Impulse control behaviours (ICBs) in Parkinson's disease (PD) are common, but meta-analytical data on their prevalence and synthesised evidence for ICB treatments in PD are significantly outdated, necessitating an update that integrates newer studies. We systematically reviewed empirical studies to update the prevalence of ICBs in idiopathic PD and narratively synthesise the efficacy of existing therapeutic interventions.

Methods

We searched PubMed, Web of Science, EMBASE (Ovid), MEDLINE (Ovid), and the Cochrane Library from inception to June 1, 2023, including quantitative studies on human participants with idiopathic Parkinson's disease. Risk of bias was evaluated using the Joanna Briggs Institute tool. The primary outcome measures were ICB prevalence and ICB changes following psychological, pharmaceutical, or brain-based interventions (Registry: Open Science Framework, 10.17605/OSF.IO/ASG2N).

Results

Thirty-seven studies were analysed. A random-effects meta-analysis of 27 studies (n = 17,906) found a mean event rate for at least one ICB in PD of 0.19 [95% CI = 0.15, 0.24], with evidence suggesting higher rates in patients taking dopamine agonists. A narrative synthesis of 10 studies (n = 283) found preliminary evidence for the effectiveness of cognitive behavioural therapy, levodopa infusion, atomoxetine, naltrexone, valproic acid, and deep brain stimulation in supporting ICB reduction.

Conclusions

ICBs represent a substantial comorbidity in PD. While several intervention strategies show promise, the paucity of high-quality treatment studies requires findings to be interpreted with caution. Clinical screening and large-scale tracking of ICBs in PD are recommended, alongside rigorous future trials.

Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder affecting 1% of people over 60 (1). While traditionally defined by clinical motor symptoms such as bradykinesia and resting-state muscular rigidity, PD is increasingly recognized as a complex neuropsychiatric condition due to the severe psychiatric symptoms that often accompany the disease (2). Within this framework, impulse control behaviours (ICB) have garnered particular attention. The purpose of this review was therefore to provide updated meta-analytical evidence of the prevalence of ICBs in PD and a narrative synthesis of the evidence for ICB treatments in PD.

Prevalence of Impulse Control Behaviours

While Impulse Control Disorders (ICDs) refer to strict, categorical psychiatric diagnoses (such as pathological gambling), Impulse Control Behaviours (ICBs) represent a broader clinical spectrum. This spectrum encompasses both formal ICDs and subsyndromal, PD-specific phenomena, such as punding and hobbyism, that may not meet rigorous

psychiatric criteria but still profoundly disrupt a patient's daily functioning and quality of life. ICBs cover pathological gambling, hypersexuality, compulsive buying, compulsive eating, *hobbyism*, punding (i.e., the frequent repetition of non-goal-oriented behaviours), compulsive medication use, hoarding, and walkabout. Prevalence rates of ICBs in PD, as reported in existing literature, exhibit considerable variability, spanning from 14% to 40%, highlighting the importance of better understanding ICBs in PD (3). The most recent meta-analysis conducted in 2018 of studies looking at the prevalence of ICBs in PD included 14 case-control studies (4). This synthesis study reported that the estimated odds ratio (OD) of having at least one ICB (of any kind) in PD compared to healthy controls was 2.07. Moderator analyses showed that usage of PD medication and disease duration were significant predictors of increased ORs for comorbid ICBs. The authors noted that their results should be interpreted cautiously given the impact of potential publication bias and the impact of the small study effect. Studies with smaller sample sizes have a relatively higher risk of overestimating the reported effects with larger effects, in turn, being more likely to be published than smaller or null effects. As the principles of open science continue to become more widespread, more recently published literature may reflect more reliable findings, highlighting the potential utility of an updated meta-analysis. Furthermore, there is evidence that ICB prevalence and most reported subtypes vary as a function of culture, socio-economical, and political influences (5). Given behavioural norms across cultures shift over time, alongside recent evolutions in clinical practice, such as shifts in dopamine agonist prescribing guidelines and the increased use of device-aided therapies, establishing a modernized, highly powered prevalence baseline is critically needed to guide current clinical screening protocols.

Treating Impulse Control Behaviours

While traditional therapeutic interventions for PD have predominantly addressed the motor symptoms, the unmet need pertaining to the non-motor aspects of PD remains considerable. First, those with PD and ICBs experience higher rates of comorbid depression and report more compromised quality of life compared to those without ICBs (6). Second, the administration of dopamine receptor agonists (DA) for PD motor symptoms has been linked to a 2–3.5-fold increased risk of developing ICBs compared to patients without DA treatment (7). And third, the estimated incidence of ICBs in PD patients treated with DA also appears to increase over time, with a 5-year cumulative incidence of nearly 50% (8). In addition, it is also noteworthy that patients with PD may also often underestimate the presence and severity of ICB symptoms, possibly influenced by a lack of insight, denial, and feelings of shame or guilt (9). That said, the last systematic review of ICB treatment in PD was conducted over a decade ago, meaning that an updated synthesis would have clear clinical value for helping people living with PD manage presentations of ICBs (10).

Objectives of the Current Review

In the current meta synthesis, we sought to review existing literature on ICBs in PD in order to achieve two main aims. The driving rationale for this review is that PD-related dopaminergic therapy drives a spectrum of hyper-dopaminergic behaviours; therefore, focusing exclusively on strict ICDs underestimates the true clinical burden. By assessing this broader umbrella of ICBs, we first aimed to update meta-analytical evidence of the prevalence of ICBs in individuals diagnosed with PD. Second, we sought to narratively synthesise evidence of the efficacy of pharmacological and non-pharmacological interventions in managing ICBs in the PD population. By mapping the existing treatment landscape, this secondary aim serves as a targeted scoping exercise to highlight the current paucity of high-quality clinical trial data and identify critical gaps requiring future rigorous investigation.

Methods

Search Strategy and Data Sources

A limited-scope search was initially conducted on Web of Science and PubMed to identify relevant index terms for the final systematic search strategy (see Table S1 in the online supplement). A single, unified systematic search was performed to identify studies on two distinct clinical outcomes: (1) the prevalence of ICBs in PD, and (2) the efficacy of ICB treatments in PD. The systematic search was performed on PubMed, Web of Science Core Library, EMBASE (Ovid), MEDLINE (Ovid), and the Cochrane Library from the databases' inception to the 1st of June 2023. We also manually searched the reference list of included studies to identify additional studies for inclusion. The systematic search and reference-list review were conducted by two independent reviewers. The finalized methodology and search strategy were retrospectively registered on the Open Science Framework to ensure methodological transparency (Registration DOI: 10.17605/OSF.IO/ASG2N).

Inclusion and Exclusion Criteria

Original studies, of any quantitative research design, on human participants, of any demographic, who had been diagnosed with idiopathic PD were included. All durations and treatment statuses of participants with PD, with or without any comorbidities, were considered eligible. The ICBs included, but not limited to, were pathological gambling, compulsive shopping, compulsive eating, hypersexuality, and punding. Studies involving atypical Parkinsonism, or which were not available as a full text written in English, were excluded.

Data Selection

Records were first imported and deduplicated in Rayyan. Two independent reviewers screened the entries with our inclusion criteria and disagreements were addressed with a third reviewer after the unblinding process. Data from the included studies were summarised in a spreadsheet that captured study details, sample characteristics, PD features (i.e., diagnostic tool, duration, cognitive status, and treatment plans), and details of any ICBs (i.e., prevalence, treatments, comparators, and baseline-follow-up effectiveness measures). Consistent with our rationale to evaluate these symptoms on a spectrum, we deliberately did not distinguish between studies that defined cases as strict impulse control disorders (e.g., diagnosed via psychiatric interview) and those that defined them as impulse control behaviours (e.g., identified via validated screening tools). Data from both assessment approaches were pooled under the broad umbrella of ICBs to capture the complete clinical burden.

Data Analysis

Where appropriate, statistical computations were conducted using Comprehensive Meta-Analysis Version 4 (11), with the remaining studies on ICB treatments being synthesised narratively. The effect size for our meta-analysis was the event rate for the prevalence of (at least one) ICB in the included studies. A random-effects model was planned a priori in order to capture between-study heterogeneity and allow for a distribution of true underlying effect sizes (compared to a single underlying effect size assumed in a fixed-effects model). Specifically, to calculate the pooled event rate, raw prevalence proportions were logit-transformed prior to analysis to stabilize the variance and ensure appropriate confidence interval estimation. The between-study variance was estimated using the DerSimonian-Laird method. We deliberately chose to pool all studies to yield a single, overarching prevalence estimate, rather than separating cohorts by dopamine-treatment status. The scientific rationale for this approach was to establish the overall global burden of ICBs across the broad, heterogeneous Parkinson's disease population. Establishing this macro-level baseline is essential for informing general, clinic-wide screening protocols, as it accurately reflects real-world clinical settings where patient medication regimens are highly individualized, frequently adjusted, and often overlap.

Quality Assessment

The Joanna Briggs Institute Critical Appraisal tool for Prevalence Studies was used by two independent reviewers to assess the studies' risk of bias (12). This appraisal tool assesses several aspects of a study's quality, in which each question can be answered *yes*, *no*, *unclear*, or *not applicable*. As the appraisal tool offers no guidance, it was decided a priori that studies would be excluded from our review if they received two *no* responses or a combination of *no* and two *unclear* responses.

Code availability

No custom computer code or algorithms were generated for this study. Statistical analyses were conducted using the commercially available Comprehensive Meta-Analysis Version 4 software.

Results

Our systematic review involved screening 2,377 unique articles, followed by an in-depth examination of 93 selected articles and the inclusion of 37 studies into the final analysis (see

Figure 1 for PRISMA selection algorithm flow diagram). Among these, 27 studies contributed data on ICB prevalence (n = 17,906 participants), which were used in our meta-analysis, and 10 studies investigated treatment options for ICBs (n = 283 participants). Sample sizes varied considerably across studies ranging from 80 to 3090 participants. For the range and overall average age, percentage of males, PD duration, and PD stage (Hoehn-Yahr scale) of participants from the included studies, see Table S2 in the online supplement.

[INSERT FIGURE 1 HERE]

Assessments of cognitive impairment were varied with some studies not reporting cognitive data, some excluding participants based on cutoff scores, and others including participants regardless of cognition. ICBs were also diagnosed through a variety of means including, DSM-based clinical interviewing, self-reported questionnaires and home-based surveys, triangulation with caregivers, and the use of medical databases. Some studies also used the Minnesota Impulsive Disorders Interview (MIDI), while most studies employed the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease (QUIP) as a base for ICB assessment. For a study-by-study summary of the studies on ICB prevalence's origin, sample size, sex, age, duration and severity of PD, and cognitive impairment measurements, see Table S2 in the online supplement.

Prevalence of Impulse Control Behaviours

Diagnosis Effects

Results suggest that ICBs are seen in both PD patient groups and healthy controls (HC). For a summary of the prevalence of at least one and two or more ICBs in HC and PD patients, see Table 1.

Table 1

Summary statistics for ICB prevalence (%) in healthy controls vs. Parkinson's disease grouped by at least one ICB and two or more ICBs.

Number of Reported ICBs	Healthy Controls (n studies)	Parkinson's Disease (n studies)	Fold Increase (PD vs. HC)
$\geq$ 1 ICB	**12.6% ± 6.7%** [4.6–20.3%]	**21.5% ± 11.1%** [5.6–45.0%]	**~ 1.7×**
$\geq$ 2 ICBs	**2.1% ± 0.7%** [1.6–2.6%]	**13.0% ± 9.9%** [0.9–32.6%]	**~ 6.2×**

Note: HC=healthy controls; ICB=impulse control behaviour; PD=Parkinson's disease; Data presented as Mean ± SD [Min–Max].

[INSERT Table 1 HERE]

Specific impulse control behaviours. There was also evidence of different prevalence of specific ICBs across PD patients and HC. For a summary of the ICB-specific prevalence in the included studies, see Table 2.

Table 2

Summary statistics for the prevalence of ICB (%) subtypes in healthy controls and Parkinson's disease patients.

ICB Subtype	Healthy Controls (%)	Parkinson's Disease (%)	Fold Increase (PD vs. HC)
Punding	**1.1 ± 1.1** [0.0–2.1]	**6.3 ± 4.8** [0.2–15.7]	~ 5.7×
Hypersexuality	**1.3 ± 1.9** [0.0–3.5]	**6.1 ± 4.7** [1.3–22.8]	~ 4.7×
Gambling	**0.9 ± 0.3** [0.7–1.3]	**3.5 ± 2.3** [0.4–7.1]	~ 3.9×
Hobbyism	**10.4 ± 2.2** [8.8–11.9]	**8.7 ± 6.4** [1.5–22.6]	0.8×
Compulsive Eating	**6.5 ± 4.1** [2.4–10.5]	**6.2 ± 4.2** [0.4–13.5]	1.0×
Compulsive Buying	**5.9 ± 4.9** [0.0–9.6]	**4.9 ± 3.2** [0.9–12.5]	0.8×
Hobbyism-Punding	—	**12.7 ± 6.7** [6.9–20.0]	N/A
Compulsive Medication Use	—	**5.1 ± 4.1** [0.0–11.3]	N/A
Walkabout	—	**1.8 ± 1.7** [0.0–5.1]	N/A

Note: HC=healthy controls; ICB=impulse control behaviour; PD=Parkinson's disease; Data presented as Mean ± SD [Min–Max].

[INSERT Table 2 HERE]

Patient Characteristics. According to one study, ICB-positive compared to ICB-negative PD patients are 3 years younger, are diagnosed with PD 4 years earlier, and have a 1-year longer disease duration (6). In this study, both groups displayed similar PD symptom severity, motor performance, and cognitive function. However, among ICB-positive patients, those with multiple ICBs exhibited more severe symptoms of depression compared to those with a single ICB. Other research similarly demonstrated that patients with ICBs exhibit higher depression scores compared to those without ICBs (13). In another study, patients with ICBs compared to ICB-free PD patients tended to be male, younger, with earlier PD diagnoses, using DA therapy, and had less dementia (14). Gender differences have also been noted in

certain subtypes of ICBs, including higher rates of hypersexuality in males (5%) compared to females (0%) and higher rates of compulsive buying in females (18.5%) compared to males (6.4%) with PD (15).

Treatment Effects

Drug Naïve Cohorts. The prevalence of ICBs in drug naïve PD cohorts differed across three studies, including 11.8% (16), 17.5% (17), and 44.3% (18). In one study, a prevalence of 17.5% was detected with the MIDI assessment but not during clinical interviewing (17). There was also evidence of a different prevalence for specific behaviours, including compulsive buying (11%), hypersexuality (11%), intermittent explosive disorder (7%) (17), and for gambling (1.2%), compulsive buying (3.0%), hypersexuality (4.2%), compulsive eating (7.1%), punding (4.8%), hobbyism (5.4%), walkabout (0.6%), and a category for *other* (18.5%) (18).

Dopamine-Levodopa Recipients. In one study, the rate of at least one ICB in participants taking DA medication (DA+) was 17.1% compared to drug naïve patients' prevalence of 6.9% (19). According to a longitudinal study, at least one ICB was seen in 26.5% (DA-/levodopa+), 26.7% (DA+/levodopa-), and 30.1% (DA+/ levodopa+) of patients: rates which stayed stable over a 2-year period (6). In another study, at least one ICB was seen in 7.2% (DA-/levodopa+), 14.0% (DA+/levodopa-), 17.7% (DA+/ levodopa+), and 1.7% (DA-/levodopa-) of patients (7).

Specific impulse control behaviours. One study reported different prevalence for specific impulse control behaviours as a function of medication status in compulsive eating (DA + = 26.6%, DA- = 17.2%), compulsive buying (DA + = 20.3%, DA- = 6.9%), hobbyism (DA + = 20%, DA- = 8%), compulsive medication use (DA + = 8.6%, DA- = 6.3%), hypersexuality (DA + = 13.3%, DA- = 6.4%), and gambling (DA + = 6.3%), DA- = 0.6%) (20).

Statistical Analyses

The mean effect size for the event rate of at least one ICB across the 24 selected studies (Table 3) was 0.19 [0.15, 0.24].

Table 3

Summary of results using the random-effects model with weighted input from included studies.

Model	Study name	Event Rate	Lower Limit	Upper Limit	Weight (Random)
	Lee et al.	**0.10**	**0.09**	**0.12**	**4.31**
	Solla et al.	0.17	0.13	0.21	4.22
	Kenangil et al.	0.06	0.04	0.08	4.13
	Weintraub et al.	0.14	0.12	0.15	4.37
	Chiang et al.	0.05	0.03	0.09	3.86
	Hassan et al.	0.22	0.17	0.26	4.24
	Lim et al.	0.24	0.18	0.30	4.16
	Kim et al.	0.16	0.12	0.20	4.18
	Joutsa et al.	0.33	0.30	0.37	4.32
	Perez-Lloet et al.	0.26	0.20	0.32	4.18
	Poletti et al.	0.08	0.06	0.10	4.25
	Callesen et al.	0.36	0.32	0.40	4.32
	Kovacs et al.	0.21	0.18	0.24	4.31
	Azmin et al.	0.11	0.06	0.20	3.53
	Zhang et al.	0.31	0.24	0.39	4.13
	Antonini et al.	0.34	0.32	0.37	4.35
	Kon et al.	0.21	0.15	0.29	4.06
	Markovic et al.	0.20	0.13	0.29	3.94
	Otmani et al.	0.28	0.21	0.37	4.08
	Vargas et al.	0.17	0.12	0.23	4.12
	Garcia et al.	0.17	0.14	0.20	4.29
	Simoni et al.	0.34	0.26	0.43	4.09
	Binck et al.	0.12	0.09	0.15	4.22
	Tolosa et al.	0.45	0.42	0.48	4.35
Random		**0.19**	**0.15**	**0.24**	
Pred Int		**0.19**	**0.05**	**0.52**	

[INSERT Table 3 HERE]

Heterogeneity Test Results

The Q-test results indicated that the across-study effect size did not exhibit uniformity (Table 4). Results of the I-squared statistics demonstrated that 97% of effect size variability came from genuine variations rather than sampling

error. Given the heterogeneity across the studies, a meta-analyses of the prevalence of the subtypes of ICBs was not possible.

Table 4
Heterogeneity test summary for fixed and random-effects models.

Model		Effect Size (95% CI)			Prediction Interval		Between Study		Other Heterogeneity Statistics			
	N	Point Estimate	Lower Limit	Upper Limit	Lower Limit	Upper Limit	Tau	Tau^2	Q	df (Q)	*p*	I^2
Fixed	24	0.22	0.21	0.23					906.80	23	< 0.001	97.46
Random	24	0.19	0.15	0.24	0.05	0.52	0.72	0.52				

Note: CI=confidence interval; df=degrees of freedom

[INSERT Table 4 HERE]

Treatment of ICBs

For a summary of the key data from studies focusing on ICB treatments, see Table S4 in the online supplement.

Psychological Interventions

In a randomised controlled trial, patients receiving cognitive behavioural therapy (CBT) for their PD-related ICBs showed decreases in clinician-rated global symptom severity scores with small-to-medium effect sizes ($\eta^2 = 0.21$) compared to patients receiving standard medical care (21). In another study, CBT, combined with standard medical care, resulted in decreased severity of ICBs, compared to standard medical care alone at 6-months follow-up. These results held true for patients with pathological gambling, both with and without comorbid PD (22).

Pharmacological Interventions

One study showed that levodopa infusion improved impulsivity and dopamine dysregulation syndrome (DDS) in PD patients, achieving symptom reduction across various types of ICBs (23). Among the five patients showing punding behaviour, improvements were observed in all cases, although complete remission of punding was achieved in only one patient. Across a series of studies on atomoxetine, the drug has been shown to improve inhibition, leading to enhancements in stopping accuracy in cognitive tasks, such as the stop signal task (Cohen's $d = 2.06$), and reductions in risk-taking behaviour (Cohen's $d = 0.25$). However, it should be noted that these studies measured proxies of cognitive impulsivity rather than changes on formal ICD clinical scales (24). In addition, atomoxetine has also been shown to restore the response inhibition network in patients with PD (25). Naltrexone has been shown to relate to a reduction in patient-rated ICB symptoms compared to placebo (Cohen's $d = 0.40$), but not in rating scales completed by clinicians (26). In a small case-control study of 4 patients, valproic acid was shown to relate to a reduction in DDS symptoms in patients with PD (27).

Other Interventions

In one study of subthalamic deep brain simulation, post-operative improvements in ICB questionnaire scores were reported in 12 of 16 patients with an effect size of Cohen's $d = 4.2$ (28). In another longitudinal study, subthalamic deep brain simulation successfully treated all pre-operative ICBs, but some patients showed de novo ICBs between 1 and 7 years after surgery (29). In one study using low-frequency repetitive transcranial magnetic stimulation, it was found that this treatment transiently suppressed punding behaviours in PD patients (30).

Discussion

The aim of the current study was to synthesise updated evidence on the prevalence of ICBs in PD, as well as evidence for the efficacy of therapeutic interventions for treating ICBs in the PD population. To our knowledge, this systematic review and meta-analysis represents the largest study of this nature conducted in the field to date, representing an update since the last synthesis of ICB prevalence in PD in 2018.

The calculated mean effect size for the prevalence of ICBs in PD, across the 24 studies included in our meta-analysis, was 19.2%. This substantial prevalence reinforces our driving hypothesis that ICBs exist on a broad clinical spectrum. By deliberately pooling data across both strict psychiatric ICD diagnoses and subsyndromal ICB screening tools, our results elucidate the true clinical burden of these hyper-dopaminergic behaviours and reveal a scope of impairment that is significantly larger than what is captured by rigid diagnostic criteria alone. The highest reported prevalence of an ICB in PD in a single study was for punding. As for healthy controls, the reported rates of at least one ICB varied between 4.6% and 15.2% with the most prevalently reported ICB in healthy controls being hobbyism (16). Among individuals with PD who had not yet been treated with medication for PD, the prevalence of ICBs ranged from 0% to 44.3% (16–18). Subgroup analyses within the reviewed studies consistently pointed to a higher prevalence of ICBs among patients treated with dopamine agonists (DA). However, the prevalence of ICBs in PD patients, whether on DA treatment or not, tended to exceed that of the general population, suggesting a predisposing vulnerability to developing ICBs in patients with PD.

While the primary driver of ICBs appears to be pharmacological, emerging evidence suggests that some patients may also possess a baseline genetic vulnerability to ICBs in PD, which may involve genes associated with the signalling pathways connected to G protein-coupled receptors (31). Given the role of dopamine in reward processing, the dopamine dysregulation hypothesis would suggest that aberrant functioning of dopaminergic neurons in the substantia nigra in PD plays a role in the emergence of ICBs (32). Exacerbated dysregulation of mesolimbic dopamine transmission associated with the use of DA medication may affect reward processing that leads to the higher prevalence of ICBs in PD patients who are DA+. It is possible that DA medication may sensitise the reward system in the basal ganglia, which leads to exaggerated reward processing, which is seen symptomatically as ICBs. Alternatively, insufficient top-down regulation of dysregulated midbrain reward processing through the recruitment of frontostriatal circuits may lead to a deficit in inhibitory mechanisms or executive function that contribute to the emergence of ICBs in PD (33).

The treatments evaluated in this review demonstrated a collective improvement in ICB symptoms, though the data remain too sparse to establish a single gold-standard approach. Instead, our synthesis suggests that treatment efficacy depends heavily on the specific intervention and the targeted ICB subtype. Neuromodulation, specifically deep brain stimulation (DBS), demonstrated the largest quantifiable benefit on pre-operative ICBs (Cohen’s d = 4.2), though its application is limited by invasive risks and the potential emergence of de novo ICBs post-surgery (28). For specific motor-loop ICBs like punding, continuous levodopa infusion showed targeted efficacy, achieving symptom reduction and partial remission by stabilizing dopaminergic stimulation. Pharmacological interventions yielded mixed results: while atomoxetine significantly improved cognitive proxies of inhibition (Cohen’s d = 2.06), it was not directly assessed against clinical ICB diagnoses (24). Naltrexone reduced patient-rated symptom severity (Cohen's d = 0.40) but failed to improve clinician-rated impressions (26). Finally, cognitive behavioural therapy (CBT) emerged as the most consistently evaluated and accessible non-pharmacological adjunct, reliably producing small-to-medium improvements ($\eta^2 = 0.21$) in global symptom severity across broad ICB presentations, including pathological gambling (21). Although promising, it is crucial to acknowledge the limited availability of high-quality studies in this field on pharmacological, psychological, and surgical interventions for ICBs. While case studies offer valuable descriptive insights, their vulnerability to selection bias significantly limits the extent to which their findings can be applied and generalised.

Moreover, an understanding of the specific ICB subtypes responding to intervention would be useful and perhaps an important focus for future research. Identifying how PD biomarkers associate with ICBs, or establishing ICB-specific biomarkers, as has been done in other literature, would help in the recruitment of patients for trials and in the evaluation of treatment efficacy for ICBs in PD (34). Consistent with previously conducted reviews on impulse control disorders, the available data remain insufficient to confidently recommend one treatment approach over another in addressing the ICBs seen in PD (4).

This meta-analysis has several limitations that should be acknowledged. First, heterogeneity across studies was notable. This variance is expected given the wide variety of assessment methods used in the literature, ranging from formal clinical interviews to self-report questionnaires, as well as natural differences in the patient populations, such as variations in disease duration and medication status. While our methodology deliberately pooled these disparate approaches to capture the full spectrum of ICBs, this overarching 19.2% prevalence is best interpreted as a broad indicator of overall clinical burden rather than a strict diagnostic rate. Second, it is possible that studies with non-significant or lower prevalence rates were underreported or unpublished in the field. Third, the majority of studies were cross-sectional, limiting our ability to make inferences about the temporal dynamics and progression of ICBs in PD. Fourth, our systematic search concluded on June 1, 2023. While this represents a comprehensive synthesis of the literature up to that point, any trials published after are not captured. Nevertheless, the massive scale of the prevalence data synthesised here ensures the baseline estimates remain robust and highly clinically relevant. Longitudinal data would be helpful for understanding this trajectory. Future research should prioritise standardised diagnostic criteria, longitudinal designs, and more detailed reporting of clinical and treatment-related variables to improve the quality of future syntheses.

Conclusions

In conclusion, beyond PD motor symptoms, the varying prevalence and diverse manifestations of ICBs within PD emphasise the critical importance of early detection and comprehensive assessment of these neuropsychiatric symptoms in PD. Clinicians should adopt a proactive approach, identifying potential ICB symptoms among PD cohorts, and perhaps scaling efforts to identify and intervene in PD-ICBs as has been done with other neuropsychiatric variables, such as cognitive impairment (35). First line management may include pharmacological intervention or deep brain stimulation for patients with severe ICBs, coupled with adjunct psychological interventions, such as CBT. CBT interventions are digitizable and can be administered by a variety of trained professionals, as well as through self-help and psychoeducational resources, potentially yielding an attractive cost-benefit balance in supporting people with PD and comorbid ICBs. Given ICBs in PD are associated with more severe symptoms of depression, understanding which cognitive mechanism relate depression, ICBs, and PD, as has been done in other neuropsychiatric diseases, would facilitate the use of CBT in this population (36). Further large and prospective studies are needed to validate the utility of such interventions in this context, and with increasing clinician recognition of ICBs in this patient cohort, such research may lead to significant patient benefit in the future by establishing a robust evidence base for treatment approaches.

Abbreviations

ICB: Impulse Control Behaviour

ICD: Impulse Control Disorder

PD: Parkinson's Disease

DA: Dopamine Agonists

Hobbyism: The excessive and dysfunctional engagement with enjoyable activities, which when done in moderation is typically pleasurable and non-pathological

Punding: The perseverative and excessive engagement with behaviours that are non-goal-directed, such as sorting, handling or rearranging objects

Declarations

Human Ethics and Consent to Participate

Human Ethics and Consent to Participate declarations: not applicable.

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Competing Interests

Dr. Reid, Ms. Makukh, Mr. Erukulla, Mr. Sargent, and Dr. Koychev report no financial relationships with commercial interests.

Author Contribution

GR: Validation, Visualisation, Writing – original draft, & Writing – review and editing. MM: Formal analysis, Investigation, & Writing – original draft. RE: Validation, & Writing – review and editing. BS: Validation, & Writing – review and editing. IK: Conceptualization, Project administration, Supervision, & Writing – review and editing.

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Data Availability

All data generated or analysed during this study are included in this published article and its supplementary information files. Additional study-level data extracted for the meta-analysis are available from the corresponding author upon reasonable request.

References

1. Tysnes OB, Storstein A (2017) Epidemiology of Parkinson's disease. J Neural Transm 124(8):901–905. 10.1007/s00702-017-1686-y
2. Koychev I, Okai D (2017) Cognitive–behavioural therapy for non-motor symptoms of Parkinson's disease: a clinical review. Evid Based Ment Health 20(1):15–20. 10.1136/eb-2016-102574

3. Kelly MJ, Baig F, Hu MT, Okai D (2020) Spectrum of impulse control behaviours in Parkinson's disease: pathophysiology and management. J Neurol Neurosurg Psychiatry 91(7):703–711. 10.1136/jnnp-2019-322453
4. Molde H, Moussavi Y, Kopperud ST, Erga AH, Hansen AL, Pallesen S (2018) Impulse-Control Disorders in Parkinson's Disease: A Meta-Analysis and Review of Case-Control Studies. Front Neurol 9:330. 10.3389/fneur.2018.00330
5. Parra-Díaz P, Chico-García JL, Beltrán-Corbellini Á, Rodríguez-Jorge F, Fernández-Escandón CL, Alonso-Cánovas A et al (2021) Does the country make a difference in impulse control disorders? A systematic review. Mov Disord Clin Pract 8(1):25–32
6. Antonini A, Barone P, Bonuccelli U, Annoni K, Asgharnejad M, Stanzione P (2017) ICARUS study: prevalence and clinical features of impulse control disorders in Parkinson's disease. J Neurol Neurosurg Psychiatry 88(4):317–324. 10.1136/jnnp-2016-315277
7. Weintraub D, Koester J, Potenza MN, Siderowf AD, Stacy M, Voon V et al (2010) Impulse Control Disorders in Parkinson Disease: A Cross-Sectional Study of 3090 Patients. Arch Neurol 67(5). 10.1001/archneurol.2010.65
8. Corvol JC, Artaud F, Cormier-Dequaire F, Rascol O, Durif F, Derkinderen P et al (2018) Longitudinal analysis of impulse control disorders in Parkinson disease. Neurology 91(3). 10.1212/WNL.0000000000005816
9. Zhang JF, Wang XX, Feng Y, Fekete R, Jankovic J, Wu YC (2021) Impulse Control Disorders in Parkinson's Disease: Epidemiology, Pathogenesis and Therapeutic Strategies. Front Psychiatry 12:635494. 10.3389/fpsyt.2021.635494
10. Callesen MB, Scheel-Krüger J, Kringelbach ML, Møller A (2013) A Systematic Review of Impulse Control Disorders in Parkinson's Disease. J Park Dis 3(2):105–138. 10.3233/JPD-120165
11. Borenstein M (2022) Comprehensive meta-analysis software. Systematic reviews in health research: meta-analysis in context. pp. 535–48
12. Munn Z, Barker TH, Moola S, Tufanaru C, Stern C, McArthur A et al Methodological quality of case series studies: an introduction to the JBI critical appraisal tool. JBI Database Syst Rev Implement Rep. 2019;Publish Ahead of Print. 10.11124/JBISRIR-D-19-00099
13. Santos-García D, De Deus Fonticoba T, Cores Bartolomé C, Suárez Castro E, Jesús S, Mir P et al (2021) Depression is Associated with Impulse-compulsive Behaviors in Parkinson's disease. J Affect Disord 280:77–89. 10.1016/j.jad.2020.11.075
14. Poletti M, Logi C, Lucetti C, Del Dotto P, Baldacci F, Vergallo A et al (2013) A single-center, cross-sectional prevalence study of impulse control disorders in Parkinson disease: association with dopaminergic drugs. J Clin Psychopharmacol 33(5):691–694
15. Kovács N, Deli G, Aschermann Z, Makkos A, Janszky J, Kovács M (2016) Impact of gender on impulse control disorders and other non-motor symptoms among Parkinson's disease patients. Parkinsonism Relat Disord 22:8
16. Marković V, Stanković I, Petrović I, Stojković T, Dragašević-Mišković N, Radovanović S et al (2020) Dynamics of impulsive–compulsive behaviors in early Parkinson's disease: a prospective study. J Neurol 267(4):1127–1136. 10.1007/s00415-019-09692-4
17. Antonini A, Siri C, Santangelo G, Cilia R, Poletti M, Canesi M et al (2011) Impulsivity and compulsivity in drug-naïve patients with Parkinson's disease. Mov Disord 26(3):464–468. 10.1002/mds.23501
18. Weintraub D, Papay K, Siderowf A, for the Parkinson's Progression Markers Initiative (2013) Screening for impulse control symptoms in patients with de novo Parkinson disease: A case-control study. Neurology 80(2):176–180. 10.1212/WNL.0b013e31827b915c
19. Lim SY, Tan ZK, Ngam PI, Lor TL, Mohamed H, Schee JP et al (2011) Impulsive–compulsive behaviors are common in Asian Parkinson's disease patients: Assessment using the QUIP. Parkinsonism Relat Disord 17(10):761–764. 10.1016/j.parkreldis.2011.07.009

20. Tolosa E, Ebersbach G, Ferreira JJ, Rascol O, Antonini A, Foltynie T et al (2021) The Parkinson's Real-World Impact Assessment (PRISM) Study: A European Survey of the Burden of Parkinson's Disease in Patients and their Carers. J Park Dis 11(3):1309–1323. 10.3233/JPD-212611
21. Okai D, Askey-Jones S, Samuel M, O'Sullivan SS, Chaudhuri KR, Martin A et al (2013) Trial of CBT for impulse control behaviors affecting Parkinson patients and their caregivers. Neurology 80(9):792–799. 10.1212/WNL.0b013e3182840678
22. Jiménez-Murcia S, Bove FI, Israel M, Steiger H, Fernández-Aranda F, Álvarez-Moya E et al (2012) Cognitive-Behavioral Therapy for Pathological Gambling in Parkinson's Disease: A Pilot Controlled Study. Eur Addict Res 18(6):265–274. 10.1159/000337442
23. Catalán MJ, Pablo-Fernández E, Villanueva C, Fernández-Diez S, Lapeña-Montero T, García-Ramos R et al (2013) Levodopa infusion improves impulsivity and dopamine dysregulation syndrome in Parkinson's disease. Mov Disord 28(14):2007–2010. 10.1002/mds.25636
24. Kehagia AA, Housden CR, Regenthal R, Barker RA, Müller U, Rowe J et al (2014) Targeting impulsivity in Parkinson's disease using atomoxetine. Brain 137(Pt 7):1986–1997. 10.1093/brain/awu117
25. Rae CL, Nombela C, Rodríguez PV, Ye Z, Hughes LE, Jones PS et al (2016) Atomoxetine restores the response inhibition network in Parkinson's disease. Brain 139(Pt 8):2235–2248. 10.1093/brain/aww138
26. Papay K, Xie SX, Stern M, Hurtig H, Siderowf A, Duda JE et al (2014) Naltrexone for impulse control disorders in Parkinson disease: A placebo-controlled study. Neurology 83(9):826–833. 10.1212/WNL.0000000000000729
27. Sriram A, Ward HE, Hassan A, Iyer S, Foote KD, Rodriguez RL et al (2013) Valproate as a treatment for dopamine dysregulation syndrome (DDS) in Parkinson's disease. J Neurol 260(2):521–527. 10.1007/s00415-012-6669-1
28. Gee L, Smith H, De La Cruz P, Campbell J, Fama C, Haller J et al (2015) The Influence of Bilateral Subthalamic Nucleus Deep Brain Stimulation on Impulsivity and Prepulse Inhibition in Parkinson's Disease Patients. Stereotact Funct Neurosurg 93(4):265–270. 10.1159/000381558
29. Kim A, Kim YE, Kim HJ, Yun JY, Yang HJ, Lee WW et al (2018) A 7-year observation of the effect of subthalamic deep brain stimulation on impulse control disorder in patients with Parkinson's disease. Parkinsonism Relat Disord 56:3–8. 10.1016/j.parkreldis.2018.07.010 PubMed PMID: 30054182
30. Nardone R, De Blasi P, Höller Y, Christova M, Tezzon F, Trinka E et al (2014) Repetitive transcranial magnetic stimulation transiently reduces punding in Parkinson's disease: a preliminary study. J Neural Transm 121(3):267–274. 10.1007/s00702-013-1100-3
31. Prud'hon S, Bekadar S, Rastetter A, Guégan J, Cormier-Dequaire F, Lacomblez L et al (2020) Exome Sequencing Reveals Signal Transduction Genes Involved in Impulse Control Disorders in Parkinson's Disease. Front Neurol 11:641. 10.3389/fneur.2020.00641
32. Weintraub D (2009) Dopamine and impulse control disorders in Parkinson's disease. Ann Neurol 64(S2):S93–100. 10.1002/ana.21454
33. Palermo S, Morese R, Zibetti M, Dematteis F, Sirgiovanni S, Stanziano M et al (2017) Impulse control disorder and response-inhibition alterations in Parkinson's disease. A rare case of totally absent functionality of the medial-prefrontal cortex and review of literature. J Adv Res 8(6):713–716. 10.1016/j.jare.2017.09.004
34. Wang C, Reid G, Mackay CE, Hayes G, Bulte DP, Suri S (2023) A systematic review of the association between dementia risk factors and cerebrovascular reactivity. Neurosci Biobehav Rev 148:105140. 10.1016/j.neubiorev.2023.105140
35. Reid G, Vassilev P, Irving J, Ojakäär T, Jacobson L, Lawrence EG et al (2024) The usability and reliability of a smartphone application for monitoring future dementia risk in ageing UK adults. Br J Psychiatry 224(6):245–251. 10.1192/bjp.2024.18

36. Bauermeister SD, Ben Yehuda M, Reid G, Howgego G, Ritchie K, Watermeyer T et al (2023) Insulin resistance, age and depression's impact on cognition in middle-aged adults from the PREVENT cohort. BMJ Ment Health 26(1):e300665. 10.1136/bmjment-2023-300665

Figures

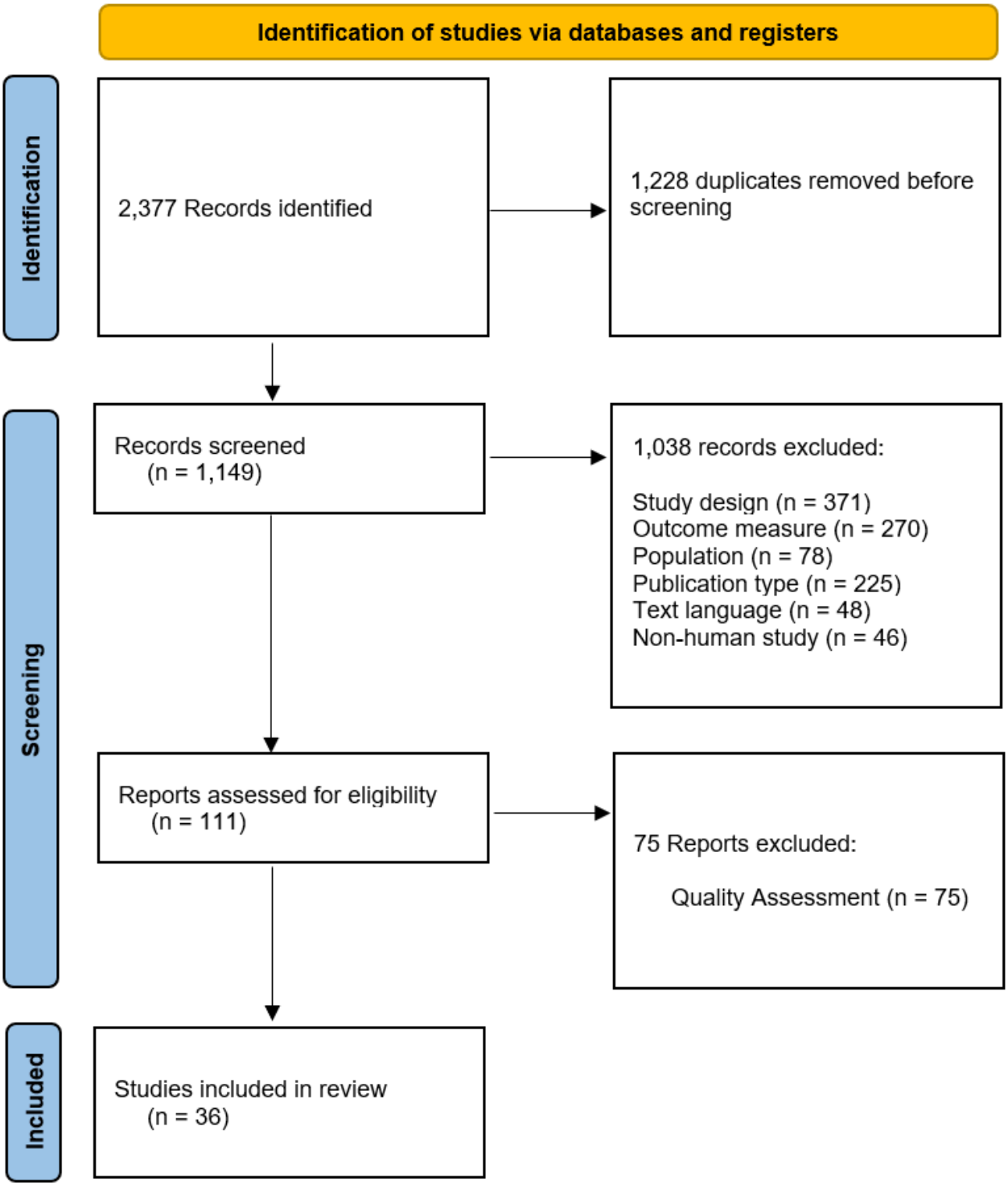

Figure 1

PRISMA flowchart of our study identification and selection process.

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