Cognitive–behavioural therapy for non-motor symptoms of Parkinson’s disease: a clinical review

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ABSTRACT

Neuropsychiatric symptoms are common in Parkinson’s disease (PD) and have a disproportionate impact on quality of life and carer burden. Pharmacological treatment is the main approach in dealing with these symptoms, but it is limited by variable efficacy and risk of drug interactions. Non-pharmacological approaches using the cognitive–behavioural therapy (CBT) model are viable alternatives and in this review paper we summarise the evidence of CBT for three of the most common psychiatric manifestations of PD: depression and anxiety, impulse-control disorders and insomnia. Most studies modified the usual CBT format to include modules accounting for problems specific to PD: activity scheduling around motoric function, motor symptoms as triggers of anxiety, fear of falling and preparation for disease progression as well as accommodation of materials for suspected executive dysfunction. We found a growing evidence base that CBT (modified to account for PD-specific problems) is effective in the treatment of PD psychiatric symptoms. Where controlled study design was used, moderate effect sizes are reported for the efficacy of CBT for depression, including with distance administration of CBT. The effects were sustained during follow-up which was between 1 and 6 months. In addition, there are some initial data on the effects of CBT on impulse-control disorders and insomnia. The studies were limited by their small and potentially unrepresentative samples and the quality of sample reporting (eg, concomitant antidepressant and dopaminergic therapy use). Additional well-designed and adequately powered studies are required to determine the utility of CBT in PD.

INTRODUCTION

Parkinson’s disease (PD) is classically defined by a triad of motor symptoms (tremor, rigidity and bradykinesia). It is, however, increasingly conceptualised as a neuropsychiatric disorder1 in view of the high prevalence of psychiatric features, including depression, impulse control disorders (ICD) and sleep difficulties.2 These non-motor complications are better predictors of disability, distress and rates to institutionalisation than the motor symptoms.3 Of the non-motor complications, mixed depression and anxiety is the most common psychiatric comorbidity, affecting ∼50% of PD patients, significantly more common that rates in the general population.4 Depression and anxiety are the strongest predictors of poorer quality of life,5 even in the advanced stages of the illness where the motor features have progressed fully.6 ICD are characterised by repetitive, reward-based behaviours, linked to the dopaminergic medication used to treat the motor symptoms. They affect ∼14% of all PD patients7 and are associated with psychiatric comorbidity and high carer burden.8 Sleep disturbance is another neuropsychiatric symptom that affects up to 60% of all PD patients.9 It is associated with somnolence10 and impairment in attention and executive function.11 On the basis of their impact on quality of life and outcome, the recognition and treatment of neuropsychiatric symptoms in PD is increasingly viewed as a priority in PD management.12

A pharmacological approach is often adopted in the management of depression, ICD and sleep disturbance in PD. In the case of depression and anxiety, the efficacy of medication was found to be of moderate effect size but statistically non-significant in a pooled analysis.13 Other issues with antidepressants include concerns over polypharmacy.14 A further complicating factor is that comorbidity with anxiety predicts lower response rate to antidepressants in PD patients.15 The usual management of ICDs relies on the withdrawal or reduction in dose of the dopamine replacement therapies. However, many patients fail to tolerate this on account of worsening control of PD symptoms, while in some the symptoms persist despite medication change.16 While there are a variety psychopharmacological treatment options for insomnia,17 their efficacy in PD is limited by its potential genesis: degeneration of sleep regulatory nervous system areas.18 Also, PD patients mainly have difficulties in sleep maintenance rather than initiation18—this is an issue as most pharmacological agents promote sleep initiation. Therefore, while pharmacological treatment of neuropsychiatric features in PD is useful and well-established, it is often limited in terms of efficacy and practicality.

The current unmet need in terms of PD neuropsychiatric therapy has led to the development of non-pharmacological approaches. The most well studied method is cognitive–behavioural therapy (CBT)—this is a brief (usually 10–12-week) talking therapy that focuses on current behaviours and problems. The underlying assumption is that when in a state of psychological distress an individual’s interpretation of everyday situations is skewed which has a negative impact on the actions they take as a result. Exploring the nature of response to individual everyday experiences, therefore, becomes the major focus of assessment and treatment. CBT aims to help people identify and subsequently modify unhelpful thoughts, and change global behaviours that serve to maintain and reinforce distorted thinking. Therefore, treatment becomes a non-didactic process of self-discovery for the patient, facilitated by the therapist, allowing opportunity for new adaptive learning and change. In other words, the patient unlearns unhelpful strategies replacing them with more adaptive behaviours. CBT has a broad evidence base in the treatment of psychiatric disorders in the general population—depression and anxiety,19 obsessive-compulsive,20 eating21 and Tourette’s disorders.22 Importantly, CBT has also been shown to be efficacious in the treatment of anxiety and depression in the context of chronic neurogenic23 and other physically disabling conditions.24

In this review paper, we aimed to provide an up-to-date summary of the evidence of psychological therapy efficacy for three of the main neuropsychiatric manifestations of PD: depression and anxiety, insomnia and impulse control disorder.

METHODS

We searched Ovid and PubMed records using search terms: ‘Cognitive-Behavioural Therapy’, ‘Behavioural’ or ‘Psychological therapy’ or ‘CBT’ and ‘Parkinson’s Disease’ or ‘PD’. Abstracts of identified articles in English were studied to select those studies that involved an efficacy evaluation or case series report of a psychological therapy for one or more of the three target symptom domains (depression and/or anxiety,
Clinical review

Evid Based Mental Health February 2017 Vol 20 No 1

Researcher

anxiety either as primary or secondary measures.25

The numbers in the controlled studies ranged from 10 to 80 patients

and problem-solving and psychoeducation.25 27 29 In addition, several

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The control groups consisted of treatment as usual or waiting lists. The larger studies included parallel carer sessions with the aim of consoli
dating learning given likely executive dysfunction.25 27 All but one

study30 reported an effect of CBT on depression had the smallest sample

size (n=6 for the CBT group) and was designed to assess the effect of

CBT for insomnia.28

The largest of the studies25 recruited 80 PD patients (mean age 64±10 years, disease duration 7±6 years) with a DSM-IV diagnosis of
depressive disorder. The participants were randomised to either CBT

plus educational programme for caregivers (n=41) or clinical monitoring

(n=39). The CBT course consisted of 10 weekly sessions and had

stronger emphasis on behavioural (behavioural activation) and anxiety

management (relaxation training, worry control) techniques as well as

sleep hygiene than usual CBT protocols. Caregivers were given 4

weekly educational sessions to help facilitate home-based CBT techni

cues practice (eg, help patients identify negative thoughts, guidance in

assisting with completion of therapy goals). The authors reported a

dependent variable. At the end of the intervention (Cohen’s d=1.59) and effect was sustained at

4-week follow-up. There was also significant improvement in several

secondary outcome measures—anxiety (HAM-A score Cohen’s d=0.98), positive reframing (COPE reframing subscale Cohen’s d=0.8), social functioning (Medical Outcomes Study Short-Form Health Survey score Cohen’s d=0.8) and severity of motor symptoms (UPDRS score Cohen’s d=0.4). There was no effect on sleep, inferences, physical dis

ability, social support or carer burden. The study is limited by the large

concomitant use of antidepressant treatment (54% of the whole sample) but their doses had to have been stable for at least 6 weeks

prior to study start.

The evidence for CBT efficacy in the treatment of depression and anxiety is supported by four uncontrolled studies30–33 and two case

series.34 35 The uncontrolled study recruited between 4 and 19 patients of similar age and disease duration to the controlled studies (table 1).

Therapy duration ranged from 6 to 14 weeks and some studies adapted

its content to limitations and problems specific to PD patients.33 The

uncontrolled studies also reported significant improvement in depressive

and anxiety at therapy end and follow-up (two studies followed partici

pants at 4 weeks30 32 and one at 24 weeks33). The two case series

(three and five patients) also reported significant improvement for
depressive symptoms,34 35 particularly for those with more severe

depression at baseline.34

RESULTS

Evidence for individual psychotherapy for depression and anxiety

We identified five controlled studies which reported effects of individual

(i.e., not group-based) psychological intervention on depression and/or

anxiety either as primary or secondary measures.25–29 Table 1 presents

an overview of the findings. In addition, there were four uncontrolled

studies and two case series.

The numbers in the controlled studies ranged from 10 to 80 patients

who had disease duration of between 5 and 10 years. The CBT courses

consisted of 6–12 weekly sessions and modified the typical CBT regime

to include a stronger emphasis on relaxation modules, behavioural

activation, problem-solving and psychoeducation.25 27 29 In addition, several

studies included modules specific to PD: activity scheduling around

on-off effects, motor symptoms as triggers of anxiety, fear of falling

and preparation for disease progression.27 29 In some instances, there

were further modifications to the CBT protocol to accommodate the

executive and somatic features of PD: less written material provided to

patients, unlimited breaks during session to allow patients to attend to

their needs.29 CBT were delivered by psychologists as well as in some

cases by master students supervised by the main investigator.29 The

control groups consisted of treatment as usual or waiting lists. The larger studies included parallel carer sessions with the aim of consoli
dating learning given likely executive dysfunction.25 27 All but one

study30 reported that CBT is substantially beneficial for anxiety and depressive symptoms as recorded through self-reported scales (see

table 1 for outcome measures and effect sizes where reported). The effect was generally evident immediately at trial end as well as at

follow-up where this had been done (between 1 and 6 months). CBT

either had lesser or no effect on broader measures of functioning as

well as carer burden (see table 1 for more details). The one study that
did not find an effect of CBT on depression had the smallest sample

size (n=6 for the CBT group) and was designed to assess the effect of

CBT for insomnia.28

PD is associated with major carer burden (copying with non-motor symp

toms; responsibility for mobility, financial and medical affairs) which can

lead to loss of quality of life through restriction of other areas of the
carer’s function.41 The CBT model has been used to provide support for

carers of PD patients. One group led on the creation of a standardised

education programme for patients with PD and their carers.42–44 The

format involved 8 weekly group sessions of 90 min duration and was

implemented across a number of European sites. The content was the

same for patients and carers and included psychoeducation sessions,
as well as core CBT modules such as relaxation training, cognitive

restructuring and situational behaviour analysis. The final sample con

sisted of 137 carers (they were not selected on the basis of psycholog

ical need) and 151 patients.42 43 The studies found that the intervention

was associated with high degree of acceptability to carers and patients

and there was improvement in the psychosocial burden experienced by both groups. Depression scores improved post session

(determined using a visual analogue scale) but there was no significant

change in depressive self-reported scores at the end of the intervention

(there was no follow-up data). A different research group targeted
carers experiencing distress (based on General Health Questionnaire 28

(GHQ-28) score) and randomised them to CBT (n=15) or treatment

as usual.46 The CBT course lasted for a mean of 15 sessions

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CBT for carers

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(GHQ-28) score) and randomised them to CBT (n=15) or treatment

as usual.46 The CBT course lasted for a mean of 15 sessions

and followed a classical CBT module structure (challenging negative

thoughts and feelings, maladaptive rules; relaxation training; pleasant

activity scheduling) plus psychoeducation on the nature of PD and the

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### Table 1 Study characteristics

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants and primary outcome</th>
<th>Design</th>
<th>Demographics of treatment groups (mean years, SD)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cole and Vaughan (2005)</td>
<td>5 PD</td>
<td>Primary outcome: GDS</td>
<td>Individual CBT case series Duration: 7 weeks; follow-up: 8 weeks</td>
<td>Age 73.6±13.4 Disease duration 3.3 ±1.9</td>
</tr>
<tr>
<td>Dobkin et al (2006)</td>
<td>Three PD and three carers</td>
<td>Primary outcome: HAM-D</td>
<td>Individual CBT and caregiver case series Duration: not given; follow-up: 4 weeks</td>
<td>Age 62.7±12.2 Disease duration 5 ±4</td>
</tr>
<tr>
<td>Dobkin et al (2007)</td>
<td>15 PD and 15 carers</td>
<td>Primary outcome: HAM-D</td>
<td>Uncontrolled 10–14-week CBT plus educational programme for caregivers Duration: 10–14 weeks; follow-up: 4 weeks</td>
<td>Age 64.2±9.6 Disease duration 5.1 ±3.2</td>
</tr>
<tr>
<td>Dobkin et al (2011)</td>
<td>80 PD</td>
<td>Primary outcome: HAM-D</td>
<td>Controlled CBT plus educational programme for caregivers (n=41) vs clinical monitoring (n=39) Duration: 10–14 weeks; Follow-up: 4 weeks</td>
<td>Age 63.7±9.9 Disease duration 6.5 ±5.5</td>
</tr>
<tr>
<td>Dreissig (1999)</td>
<td>79 PD</td>
<td>Primary outcome: PROGRESSOR 230</td>
<td>Controlled self-help and CBT (n=9) vs treatment at usual (n=70) Duration: 12 weeks (6 sessions); Follow-up: none</td>
<td>Age 52.7±unknown Disease duration 8.6 ±unknown</td>
</tr>
<tr>
<td>Farabahag et al (2010)</td>
<td>8 PD</td>
<td>Primary outcome: HAM-D</td>
<td>Uncontrolled CBT case series Duration: 12 weeks; follow-up: none</td>
<td>Age 63.5±7.5 Disease duration unknown</td>
</tr>
<tr>
<td>Fenney et al (2005)</td>
<td>4 PD</td>
<td>Primary outcomes: BDI and STAI-S</td>
<td>Uncontrolled CBT case series Duration: 8 weeks; follow-up: 12 weeks</td>
<td>Age 65.3±11 Disease duration 4.8 ±4.9</td>
</tr>
<tr>
<td>Shimizu et al (2015)</td>
<td>19 PD</td>
<td>Primary outcome: HAM-D</td>
<td>Uncontrolled CBT study Duration: 6 weeks; follow-up: 12 weeks</td>
<td>Age 63.8±9.9 Disease duration 6.4 ±3.5</td>
</tr>
<tr>
<td>Troeung et al (2014)</td>
<td>18 PD</td>
<td>Primary outcome: DASS-21</td>
<td>Controlled CBT (n=11) vs waitlist (n=7) Duration: 8 weeks; follow-up: 16 weeks</td>
<td>Age 68±7.7 Disease duration 5.7 ±5.5</td>
</tr>
</tbody>
</table>

### Depression and anxiety (distance CBT)

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants and primary outcome</th>
<th>Design</th>
<th>Demographics of treatment groups (mean years, SD)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobkin et al (2011)</td>
<td>21 PD</td>
<td>Primary outcome: HAM-D</td>
<td>Uncontrolled telephone CBT plus caregiver educational sessions Duration: 10 weeks; follow-up: 4 weeks</td>
<td>Age 65.9±9.4 Disease duration 7.5 ±9.4</td>
</tr>
<tr>
<td>Kraepelin et al (2015)</td>
<td>9 PD</td>
<td>Primary outcome: HADS</td>
<td>Uncontrolled internet-based CBT Duration: 12 weeks; follow-up: None</td>
<td>Age 66±11.6 Disease duration 8.1 ±3.8</td>
</tr>
<tr>
<td>Veazey et al (2009)</td>
<td>10 PD</td>
<td>Primary outcomes: PHQ-9 and BAI</td>
<td>Controlled telephone CBT (n=5) vs support group (n=5) Duration: 8 weeks; follow-up: 4 weeks</td>
<td>Age 86±9.9 Disease duration unknown</td>
</tr>
</tbody>
</table>

**Table continued...**
Table 1

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants and primary outcome</th>
<th>Design</th>
<th>Demographics of treatment groups (mean years, SD)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jimenez-Murcia et al (2012)</td>
<td>15 PD and 45 non-PD pathological gamblers</td>
<td>Primary outcomes: SDGS</td>
<td>Age 62.7±8.5 disease unknown</td>
<td>The authors found higher rate of relapse (25% vs 11%) and drop-out from therapy (29% vs 9%) for the PD relative non-PD pathological gamblers. However, this difference did not reach statistical significance.</td>
</tr>
<tr>
<td>Insomnia</td>
<td>Rios Romanets et al (2013)</td>
<td>18 PD</td>
<td>Age 65.4±6.3 disease duration 5.2 ±1.8</td>
<td>Significant improvement in insomnia severity (ISI) but not SDOPA sleep score (doxepine was better than placebo for both). Examiner-rated but not participant-rated CGI improved. No change to daytime sleepiness, BDI, sleep hygiene, dysfunctional sleep beliefs or cognition. No improvement in depression scores with CBT.</td>
</tr>
<tr>
<td>Carer distress</td>
<td>Yang and Petrini (2012)</td>
<td>25 PD</td>
<td>Age 65.2±unknown disease duration unknown</td>
<td>Improvement in PDSS and sleep diary measures (onset latency, wake after sleep onset, total sleep time, time in bed and sleep efficiency).</td>
</tr>
<tr>
<td>A’Campo et al (2010)</td>
<td>151 PD and 137 carers</td>
<td>Group educational programme with CBT elements for patients and caregivers</td>
<td>Age 64.4±9.2 disease duration 6.5 ±4.3</td>
<td>Patients: improvement in psychosocial burden and visual-analogue scale depression score but no improvement in quality of life or self-reported depression. Carers: improvement in psychosocial burden and visual-analogue scale mood scores but no change to health state and self-reported depression.</td>
</tr>
<tr>
<td>Macht et al (2007)</td>
<td>151 PD</td>
<td>Group educational programme with CBT elements for patients</td>
<td>Age 64.4±9.2 disease duration unknown</td>
<td>67–80% felt intervention was appropriate and fulfilled expectations. Significant improvement in mood (visual-analogue scale) and psychosocial problems. No change to quality of life and self-rating depression score</td>
</tr>
<tr>
<td>Secker and Brown (2005)</td>
<td>30 carers</td>
<td>Controlled study of session CBT</td>
<td>Age 59.1±12.2 disease duration: not applicable</td>
<td>Reduction of psychological distress (total GHO-28 scores) for the treatment group at intervention end. The CBT group also had a decrease in somatic, anxiety, insomnia, social dysfunction but not depression GHO-28 scores (as well as GDS scores). Effect was maintained at 12-week follow-up.</td>
</tr>
<tr>
<td>Simons et al 2006</td>
<td>22 PD and 14 carers</td>
<td>Group educational programme with CBT elements for patients and caregivers</td>
<td>Age 65±7.3 disease duration 7.6 ±5.0</td>
<td>Patients and carers approved of the programme and improved in terms of visual-analogue mood scores. No significant differences for carers’ quality of life, psychosocial problems, self-reported depression.</td>
</tr>
</tbody>
</table>

CBT for impulse-control disorder in PD

Two studies to date have evaluated the efficacy of CBT in the treatment of ICD in PD.27 47 In the larger study, the authors recruited 45 PD patients, randomising 28 of them to a modified CBT programme (mean age 59±8 years, disease duration 11±6 years) and 17 to a control waiting list. The CBT intervention consisted of 12 sessions (mostly at patient’s homes) and the part relevant to ICD included psychoeducation (focusing on ICD but also on executive dysfunction in PD), motivational interviewing to identify where the patient was in terms of the cycle of change, monitoring of behaviour, problem solving and pleasant activity scheduling. The CBT for patients was augmented with a 4-week carer intervention that aimed at psychoeducation, supporting collaborative problem-solving and recognising organic personality change. The study found reduction in the global level of ICD symptom severity, their frequency and impact. At the 6-month follow-up, 44% of the treatment group no longer met ICD criteria compared with 29% of the waiting list group. While there was no significant benefit to carer burden or distress, carers did improve in terms of their levels of anxiety and depression. A smaller, retrospective study reported on the efficacy of CBT in treating pathological gambling in PD (n=15) versus non-PD (n=45) patients.45 The CBT schedule was adapted in the PD group to focus on PD psychoeducation, planning of alternative gratifying activities (eg, interactions between gambling and timing of dopaminergic drug therapy), coping with other impulse control behaviours. The investigators offered 16 weekly sessions. The authors did not find a difference between the PD and non-PD patients in terms of their treatment response (measured using South Oaks Gambling Screen46) but there was an indication of higher relapse and therapy drop-out rates among the PD patients. The study was limited by its retrospective nature, convenience of sample (clinic attendees) and lack of concomitant medication reporting.

CBT for insomnia in PD

Two studies evaluated CBT targeting insomnia in PD.28 49 The first study involved 6 weekly face-to-face CBT sessions plus light therapy compared to doxepin and placebo light therapy (each group included 6 patients, mean age 65 with 5 years disease duration).28 The second
study evaluated four-session telephone-based CBT in 22 patients (mean age 65, most participants had a disease duration of 1–5 years). Neither study reported modifications of classical CBT protocols for insomnia. The individual CBT intervention was reportedly beneficial in terms of the sleep quality index relative to doxepine and placebo, but there was no effect on day time sleepiness. The telephone-based intervention reported improvement on total sleep duration, sleep-onset latency and sleep efficiency posttreatment and at 3-month follow-up.

DISCUSSION

The aim of this review was to evaluate the level of evidence for cognitive–behavioural therapy for three non-motor symptoms in PD: depression and anxiety, ICD and insomnia. We identified a range of controlled, uncontrolled and case series evaluating CBT for depression and anxiety in PD. CBT protocols were frequently modified to account for psychological problems that are more prevalent in PD—anxiety modules were prioritised with a focus on relaxation techniques, recognising motor symptoms as triggers of anxiety and the highly prevalent fear of falling. Loss of motivation is also highly prevalent leading to a number of CBT protocols, including behavioural activation modules with emphasis on activity scheduling around on/off periods. Some authors accounted for the potential executive dysfunction in PD and amended their CBT materials to ease comprehension. The largest study offered supplemental caregiver educational programmes which was intended to reinforce learning in the patients’ environment given their expected executive and memory dysfunction.

Overall, there was consistent evidence for moderate improvement in self-reported depressive and anxiety scores post-treatment and at follow-up assessments of up to 3 months. There are a number of potential mechanisms for this improvement, including improved insight and self-reflection. Most protocols focused on self-monitoring and the beneficial effect could thus be due to improved ability to identify triggers of low mood or anxiety symptoms. Furthermore, the CBT model focuses on improving patients’ problem-solving and coping with stress skills. Also, social isolation is a significant factor in patients with PD on accounts of physical disability or perceived stigma. The psychological intervention therefore could have been beneficial through reduction of social isolation. In addition to these considerations, it is likely that the adaptations of the CBT protocol to the requirements for PD were significant factors. PD features a number of severe physical symptoms that are more prevalent in PD—motor symptoms as triggers of anxiety and the highly prevalent fear of falling. These data suggest that treatment of PD carer is most likely to be effective where systematic CBT is applied.

Limited evidence exists also for targeted treatment of insomnia and ICD. Two small studies reported beneficial effect to sleep quality from CBT using face-to-face and telephone CBT. The second largest evaluation of CBT in PD focused on ICD and found good evidence for improvement in impulse control behaviours versus waiting list. A smaller study explored CBT efficacy for pathological gambling but was limited by its retrospective and small sample size that was recruited from a convenience population (clinical patients).

Limitations

Many of the studies included in this review are small and speculative, and essentially present pilot data. The conduct of many of the constituent studies is poor, with incomplete attention paid to participation rates, gaining representative samples of consecutive patients (instead of convenience samples) and small sample sizes. The reporting of many of the studies is also poor, with insufficient attention paid to descriptions of samples, poor reporting of ‘negative’ associations and failure to report effect sizes. Follow-up periods tended to be brief which limits the extent to which conclusions can be drawn about the long-term efficacy of CBT in PD. Furthermore, concomitant antidepressant and dopaminergic medication use was either significant or unreported, which suggests a potential confound. Nonetheless, there is a small body of better quality studies, and for some of the main findings there is consistency of results within the literature. Finally, depression/anxiety, ICD and insomnia do not exhaust the range of neuropsychiatric symptoms in PD—psychosis and apathy are prominent, significant and undertreated symptoms of the disease. We chose not to focus on these domains in our review on account of the limited existing evidence for their treatment with CBT.

Conclusion

Psychiatric manifestations in PD are a highly prevalent component of the disease that is a significant source of disability and carer burden. Psychopharmacological approaches are limited in the extent to which they can consistently ameliorate them. In this review paper, we found that there is a growing evidence base for non-pharmacological treatment of psychiatric symptoms with moderate effect sizes reported for the efficacy of CBT on depression, including where distance administration was used. In addition, there is some initial promising data on the effects of CBT on impulse-control disorders and insomnia. There is a need for adequately powered studies to strengthen the position of CBT as a viable treatment option for psychiatric symptoms in PD.

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