

RESEARCH ARTICLE

Paradoxical intention for insomnia: A systematic review and meta-analysis

Markus Jansson-Fröjmark¹  | Sven Alfnsson¹ | Benjamin Bohman¹ |
Alexander Rozental^{1,2} | Annika Norell-Clarke^{3,4} 

¹Department of Clinical Neuroscience, Centre for Psychiatry Research, Karolinska Institutet & Stockholm Health Care Services, Region Stockholm, Stockholm, Sweden

²Great Ormond Street Hospital Institute of Child Health, UCL, London, UK

³Department of Social and Psychological Studies, Karlstad University, Karlstad, Sweden

⁴Faculty of Health Sciences, Kristianstad University, Kristianstad, Sweden

Correspondence

Markus Jansson-Fröjmark, Centre for Psychotherapy Education and Research, Liljeholmstorget 7B, 117 63 Stockholm, Sweden.

Email: markus.jansson-frojmark@ki.se

Summary

Paradoxical intention (PI) has been considered an evidence-based treatment for insomnia since the 1990s, but it has not been evaluated with modern review techniques such as meta-analysis. The present study aimed to conduct the first systematic review and meta-analysis of studies that explore the effectiveness of PI for insomnia on insomnia symptomatology and theory-derived processes. A systematic review and meta-analysis was conducted by searching for eligible articles or dissertations in six online bibliographic databases. Randomised controlled trials and experimental studies comparing PI for insomnia to active and passive comparators and assessing insomnia symptoms as outcomes were included. A random effects model was estimated to determine the standardised mean difference Hedge's g at post-treatment. Test for heterogeneity was performed, fail-safe N was calculated, and study quality was assessed. The study was pre-registered at International Prospective Register of Systematic Reviews (PROSPERO, CRD42019137357). A total of 10 trials were identified. Compared to passive comparators, PI led to large improvements in key insomnia symptoms. Relative to active comparators, the improvements were smaller, but still moderate for several central outcomes. Compared to passive comparators, PI resulted in great reductions in sleep-related performance anxiety, one of several proposed mechanisms of change for PI. PI for insomnia resulted in marked clinical improvements, large relative to passive comparators and moderate compared to active comparators. However, methodologically stronger studies are needed before more firm conclusions can be drawn.

KEYWORDS

adults, cognitive behavioural therapy, cognitive therapy, effectiveness, paradoxical intention, sleep disturbance

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1 | INTRODUCTION

Insomnia disorder is characterised by difficulties initiating, returning to, or waking up too early from sleep, in combination with daytime consequences (worry or functional impairment) (American Psychiatric Association, 2013). Approximately 6%–10% of the population meets criteria for insomnia disorder (Morin & Benca, 2012; Ohayon, 2002), and for the sufferer, the condition is related to a number of negative effects (e.g. psychological distress, decreased daytime functioning, and more sick leave) (Baglioni et al., 2011; Daley et al., 2009; Sivertsen et al., 2009). Together with the fact that insomnia disorder tends to remain chronic if untreated (Morin et al., 2009), these findings highlight the importance of effective treatments for the condition.

During the 1970s, Ascher and Efran (1978) developed a new insomnia treatment component: paradoxical intention (PI). PI was described as instructing patients with sleep onset insomnia to try to remain awake for as long as possible, rather than to focus on trying to fall asleep. In the first case reports (Ascher & Efran, 1978), PI was delivered to patients with insomnia who remained unimproved after a behaviourally oriented treatment approach. Ascher and Efran (1978) showed that PI resulted in a rapid reduction in sleep-onset latency (SOL) among the five patients in the case series. After the first study in 1978, other trials in the 1970s also investigated the effectiveness of PI with similar results (Ascher & Turner, 1979, 1980; Turner & Ascher, 1979).

Over the years, slightly different formats of PI have been developed. While the core still was instructions to the patient to try to remain awake, three slightly different approaches emerged. Ascher and Efran (1978) described two versions of PI; one in which the patient is instructed to record pre-sleep thoughts and one in which the patient is asked to apply relaxation. A third version was to instruct the patients to keep their eyes open, while lying comfortably in bed in a dark room (Ascher & Turner, 1979).

The theoretical underpinnings of PI have also varied over time. From the start, PI was based on the notion that patients with insomnia fail to realise that sleep is an involuntary physiological process, and instead try to mobilise their full effort to fall asleep (Ascher & Efran, 1978). It was also proposed that this wilful effort would result in frustration and arousal of the autonomic nervous system, thus interfering with sleep onset. Thereby a vicious cycle has been created, in which self-monitoring, increased arousal, performance anxiety, sleep effort, and failure to fall asleep has been established. PI is believed to work by eliminating performance anxiety (Ascher & Efran, 1978). In a later conceptualisation, PI was seen in the light of the attention–intention–effort model (Espie et al., 2006), in which selective attention to threatening sleep cues, such as noise, starts the process (Harris et al., 2015). Selective attention to threatening sleep cues leads to explicit intention to sleep, which results in the inhibition of normal de-arousal. In turn, sleep intention leads to direct and indirect sleep effort, e.g. actively trying to sleep and extending bedtime. In this model, PI is viewed as an attempt to manipulate the explicit sleep intention by remaining passively awake or by giving up any direct intention to fall asleep.

The effectiveness of PI has been reviewed over the years. In the 1990s, the American Academy of Sleep Medicine (AASM) identified six studies examining PI and concluded, based on criteria developed by the American Psychological Association (Chambless & Hollon, 1998) that the treatment component is an empirically supported intervention (Morin et al., 1999). A later review by the AASM also categorised PI as a well-established treatment (Morin et al., 2006). However, it is important to underscore that the AASM, which examined research papers up until 2004, only used two databases in the search for studies, specifically investigated treatment effectiveness on night-time symptoms, did not provide detailed methodological and statistical information in the quantitative assessment of PI, and used criteria for empirically supported treatments that have since been abandoned (e.g. on the grounds that studies not showing effectiveness is not necessarily taken into account). It is thus plausible that a new review encompassing all published studies on PI might yield different results. Further, two of the current study's authors recently performed a narrative review, which concluded that PI has empirical support for insomnia (Jansson-Fröjmark & Norell-Clarke, 2018). However, the review did not quantitatively assess the effectiveness of PI and did not specifically differentiate between outcomes (e.g. night-time and daytime symptoms). A further limitation of the scientific scrutiny of PI is that no previous meta-analysis or review has attempted to examine evidence for how the effectiveness of PI might be explained. Even though it has been suggested that performance anxiety and sleep intention are the mechanisms through which PI works (Ascher & Efran, 1978; Espie et al., 2006), these notions have not been formally reviewed.

Although the effectiveness of PI has previously been reviewed quantitatively to some extent (Morin et al., 1999, 2006) and narratively (Jansson-Fröjmark & Norell-Clarke, 2018), the present study aimed to conduct the first systematic review and meta-analysis of studies that explore the effectiveness of PI for insomnia. More specifically, the purpose was to investigate the effectiveness of PI on insomnia symptomatology (night-time and daytime symptoms) and theory-derived processes (e.g. performance anxiety).

2 | METHODS

A systematic review approach was used. Meta-analytical calculations to aggregate outcome measures were also conducted. The review was pre-registered at the International Prospective Register of Systematic Reviews (PROSPERO) in July 2019 and can be accessed at www.crd.york.ac.uk/PROSPERO/display_record.php?RecordID=137357.

2.1 | Search strategy

An extensive database search was conducted in October 2018 and June 2021 by three project-independent librarians at Karolinska Institutet University Library, Stockholm, Sweden to identify all

studies that evaluated the effectiveness of PI for insomnia. The search was carried out by using six online bibliographic databases (i.e. the Medical Literature Analysis and Retrieval System Online [MEDLINE, Ovid], Psycinfo [Ovid], Excerpta Medica dataBASE [EMBASE, Elsevier], Cumulative Index for Nursing and Allied Health Literature [CINAHL, Ebsco], Web of Science [Clarivate], and Dissertations and Theses [ProQuest]). The search strategies were developed by the three librarians in collaboration with the first author. The strategies were based on several Medical Subject Headings (MeSH) and keyword search terms. Across the databases, terms were used to identify studies in which individuals with insomnia had been included and PI had been employed. For a detailed description of the search strategies, see the Supplementary Material (Table S1). Also, the first author reviewed the reference lists of recent reviews and meta-analyses of the effectiveness of cognitive behavioural therapy for insomnia (CBT-I), as well as the reference lists of each study included in the present review.

2.2 | Selection procedure

As can be seen in Figure 1, the database search yielded a total of 214 records from the six databases, out of which 77 titles were

duplicates. Thus, 137 records were the focus for further review. The inclusion criteria for the studies in the present review were:

- The study was a randomised controlled trial (RCT) or used an experimental design, from which outcome data per group could be extracted.
- The study reported treatment outcomes using at least one outcome assessing insomnia symptomatology (i.e. night-time [e.g. SOL and sleep quality] and daytime symptoms [tiredness and depressive symptoms]).
- PI was tested as a single component in at least one group.
- The participants were adults (i.e. aged ≥ 18 years).
- The participants had been diagnosed with insomnia (no matter if it was defined as insomnia disorder or primary, secondary, or comorbid insomnia), reported undiagnosed problems with initiating or maintaining sleep, or reported poor sleep (e.g. scoring above a cut-off on a validated insomnia scale).
- The study was published before or in October 2018.
- The study was published in English.

Concerning the population criterion (e), we included studies in which participants were defined as having insomnia symptoms or poor sleep (i.e. not fulfilling all criteria for insomnia disorder) due to the growing evidence for, and trend in diagnostic systems towards,

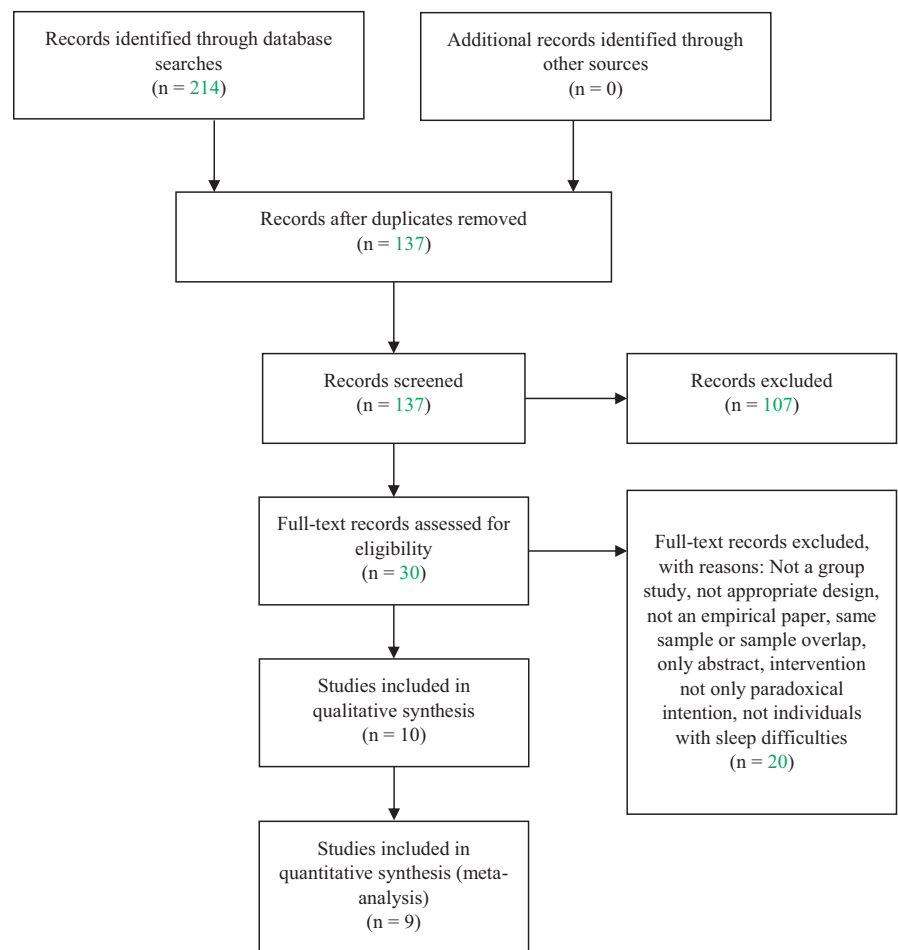


FIGURE 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) study inclusion flow chart

a dimensional view of psychopathology (Hankin et al., 2005; van Os et al., 1999). A further reason to allow inclusion of participants without diagnosed insomnia disorder is that psychological treatments (e.g. CBT-I) have been shown to effectively reduce insomnia symptomatology among those with subthreshold insomnia (Denis et al., 2020), insomnia symptoms (Swift et al., 2012), and acute insomnia (Randall et al., 2018). Also, sensitivity and moderator analyses were planned a priori, in case there was substantial heterogeneity concerning the population criterion.

The 137 records were first screened via a systematic review web application (<https://rayyan.qcri.org>). The abstracts of all the records were initially reviewed by the first and last authors (MJF and ANC) to exclude irrelevant studies. When it was unclear whether a record met the criteria through the abstract screening, the record was read in full text by the first and last authors. These authors discussed disagreements until a negotiated conclusion was reached. In total, 107 records were excluded based on the inclusion criteria described above in the abstract screening phase. Full texts of the remaining 30 references were reviewed by the same authors. In this phase, the same inclusion criteria described above were used. In total, 10 records were included at this stage. The excluded 20 studies with reasons for exclusion are listed in the Supplementary Material: Table S2.

2.3 | Data extraction

At this stage, the 10 records were reviewed by the second and the third authors (SA and BB). The raters used a standard extraction sheet to summarise information about each study: country of origin, publication type, study design, sampling (community sample, clinical sample, or other), sample size, diagnosis (i.e. insomnia disorder/primary insomnia/secondary insomnia/comorbid insomnia, insomnia symptoms [not fulfilling all criteria for a diagnosis], or poor sleep [no evidence of insomnia symptoms]), psychiatric and somatic comorbidity, mean age of participants, percentage of women, treatment-related parameters for PI and other active treatments (content, number and duration of sessions, administration, manual, therapists, training, supervision, and treatment integrity), outcome measures (i.e. insomnia-related night-time [subjective or objective sleep data] and daytime symptoms [e.g. tiredness], as well as theory-driven processes [e.g. performance anxiety]), and findings. Data extraction was accomplished independently and discrepancies between the two raters (on average 6%) were resolved through discussion with the first author (MJF).

2.4 | Assessment of study quality

The quality assessments of the included studies were indexed using standardised criteria (Kmet et al., 2004) by the second and the third author (SA and BB). The quality assessment focussed on

the extent to which design, conduct, and analyses minimise errors and biases for RCTs and experimental designs. Using a 3-point rating scale (yes = 2, partially = 1, and no = 0), the assessment involved 14 items (objective, design, method, subjects, intervention, blinding of investigators to intervention, blinding of subjects to intervention, outcome, sample size, analytical methods, variance, confounds, description of results, and conclusion). One item (blinding of investigators to intervention) was removed as this was not relevant to the present review. A global score and a percentage were calculated for each study, enabling comparisons across trials. To increase the reliability of the quality scores, the quality ratings were conducted independently by the two authors. Discrepancies between the two authors (on average 11%) were discussed until a final score was agreed upon for each study. The items are consistent with the recommendations of the Centre for Reviews and Dissemination for systematic reviews (Akers et al., 2009) and with previous study assessment approaches in the area of insomnia disorder (Miller et al., 2014; Morin et al., 2006).

2.5 | Data analysis

A meta-analysis was performed with data from the systematic review. The standardised mean differences at post-treatment, divided by the pooled standard deviations (*SDs*), were used to calculate the aggregated effects (Hedge's *g*; see below) between PI and comparators. Because comparators varied in nature, separate meta-analyses were conducted for those that were considered active (attention-control placebo, desensitisation, stimulus control, imagery, information, progressive relaxation, and feedback) and passive (waitlist and no treatment). In addition, given that some trials included several comparators of the same type (i.e. active or passive), these were combined into one comparator by summing the number of participants and pooling means and *SDs*. Furthermore, as several outcomes were used in the trials, separate meta-analyses per outcome were conducted. In addition, 95% confidence intervals (*CI*s) were included for each of the aggregated effects. Similar to Cohen's *d*, a between-group effect size Hedge's *g* can be interpreted accordingly, 0.20–0.49 are considered to represent a small effect, 0.50–0.79 a medium effect, and >0.80 a large effect (Cohen, 1988). However, per recommendations (Cumming & Finch, 2001), effect sizes were compared to other relevant estimates in the literature in order to make sense of their importance. Furthermore, a test of heterogeneity was conducted to investigate between-study variation, using the I^2 -statistic (25%, 50%, and 75% indicating low, medium, and high heterogeneity, respectively), and the *Q*-statistic to determine if heterogeneity was significant (Borenstein et al., 2011). Given that the I^2 -statistic is often regarded as imprecise, especially if the number of studies is low, 95% *CI*s were also calculated for the I^2 . Moreover, in select cases, Forest plots were produced to display the between-group effect sizes for each study and the overall benefits of PI for insomnia. Potential risk of publication bias was determined using funnel plots and Egger's tests (Egger et al., 1997). In addition, a fail-safe *N* was used to determine the number of studies having a null result that are necessary to increase the *p* value for the aggregated effect sizes

>0.05, using the Rosenberg-approach (Rosenberg, 2005). Statistical analyses were made using R and the “metafor” package (Viechtbauer, 2010), implementing a random-effects model as between-study variation was expected.

3 | RESULTS

3.1 | Systematic review

After the screening process, 10 studies were included in the review (Table 1). While eight papers were published in peer-reviewed journals, two studies were presented in dissertations (Buchanan, 1988; T. M. Byrne, 1983). Concerning country of origin, eight trials were carried out in North America and two in Europe. In terms of design, all studies, except one (Byrne, 1985), were RCTs. Nine of the trials compared PI with a passive condition (i.e. waitlist or no treatment) and eight included an active comparator (i.e. attention placebo, quasi-desensitisation placebo, stimulus control, imagery relief placebo, information, progressive relaxation, and feedback).

All studies recruited participants from the community. The sample sizes in the studies ranged from 16 to 70 (total sample size = 384). The insomnia symptomatology varied; in three studies, the participants were classified as meeting diagnostic criteria for primary insomnia. In the remaining trials, the individuals were categorised as reporting sleep-onset insomnia (six studies) or insomnia (one study). None of the studies assessed comorbidity using a structured procedure. The included participants' age and gender varied across trials; the mean age ranged from 25 to 45 years, and the majority of the participants were women (55%–67%), estimations based on trials in which data was available.

All studies used sleep diary outcomes, consisting of items assessing SOL, difficulty falling asleep, number of awakenings (NAW), total sleep time (TST), sleep efficiency (SE), effort to sleep, sleep enjoyment, and restedness. Three studies reported on objective sleep outcomes (SOL and SE) (Broomfield & Espie, 2003; Byrne, 1983; Fogle & Dyal, 1983). Four of the trials reported on questionnaire outcomes, assessing daytime symptoms, such as anxiety and depressive symptoms (Espie et al., 1989) and sleep performance anxiety (Broomfield & Espie, 2003; Buchanan, 1988; Fogle & Dyal, 1983). None of the studies reported on sleep intention as a potential treatment mechanism or on adverse effects of PI.

Concerning study quality, the mean (*SD*, range) total score across the 10 studies was 18.7 (1.7, 15–20) points out of 26. The two most common methodological limitations were: (1) when blinding of subjects would have been possible, it was not reported, and (2) the sample size was not deemed appropriate. For the ratings of each of the 13 items for each study, see Table S3.

In Table 2, the treatment-related parameters for PI are presented. The content of PI varied slightly across studies. The most common approach used the original rationale instructions for PI (Ascher & Efran, 1978). PI was delivered as individual therapy in four studies

(three to four sessions), in a self-help format in five trials (2–8 weeks), and as group therapy in one study (four sessions). Only one study reported that PI was delivered according to a manual. Report of therapist profession was either irrelevant (due to a self-help format) or rare; in the two studies in which profession was reported, PI was delivered by psychology and PhD students. Information regarding therapist training or supervision was generally not reported (for an exception see Buchanan, 1988). Only one trial checked for treatment integrity.

3.2 | Meta-analysis: insomnia symptoms

As mentioned above, the aggregated results for PI at post-treatment were separated depending on the type of comparator used, i.e. passive (Table 3) and active (Table 4). The results were also separated by outcome. The largest number of comparisons regarded SOL ($k = 7$), although the most common contrast was three. Overall, the beneficial effects for PI were larger when compared to passive rather than active comparators, with the largest being obtained for difficulty falling asleep, Hedge's $g = 1.71$ (versus a passive comparator), and the smallest for SOL, $g = 0.00$ (versus an active comparator). Relative to passive comparators, PI resulted in large improvements in SOL, difficulty falling asleep, NAW, and restedness, a moderate increase in TST, and a negligible effect size on SE. Compared to active comparators, PI showed moderate improvements in difficulty falling asleep, NAW, and restedness, and negligible effect sizes on SOL and TST. Forest plots for SOL comparing PI to passive and active comparators are shown in Figures 2 and 3.

Heterogeneity ranged from non-significant (0.00%) to high (69.14%), but no direct pattern emerged with regard to its nature. Also, because of the small number of comparisons, sub-group analyses were deemed unfeasible to perform.

As for the potential of publication bias, the Egger's test was only significant in two cases (restedness and SOL when compared to passive comparators). However, given the small fail-safe N for most outcomes (mean [*SD*] 18.18 [24.06]), few trials with a null result are required to make the aggregated effects non-significant.

3.3 | Meta-analysis: sleep performance anxiety

As shown in Table 3, quantitative data from three studies were combined to examine the effectiveness of PI versus passive comparators on sleep performance anxiety. The aggregated effect was large, $g = 1.04$, in favour of PI. Heterogeneity and the Egger's test were insignificant. Again, a small fail-safe $N = 21$ was found, indicating that few trials with a null result would turn the effect non-significant. However, performance anxiety was not possible to explore in relation to active comparators because the number of studies were too few ($k = 2$).

TABLE 1 Description of included studies examining paradoxical intention for insomnia: Design, sample description, treatment arms, outcomes, findings, and quality score

Author(s)	Design	Sample: Size, diagnosis, age (mean), % female	Treatment arms	Outcomes	Findings	Quality score (%)
Ascher and Turner (1979)	RCT	25, primary insomnia, 39 years, 60%	PI versus placebo versus no treatment	Sleep diary (SOL, NAW, restedness, difficulty falling asleep)	PI > placebo and no treatment: SOL, NAW and difficulty falling asleep.	20 (77)
Ascher and Turner (1980)	RCT	40, sleep-onset insomnia, 37 years, NR	PI versus PI with desensitisation rationale versus placebo versus no treatment	Sleep diary (SOL, NAW, restedness, difficulty falling asleep, TST)	PI > no treatment: all outcomes. PI > PI with desensitisation rationale and placebo: SOL, NAW and restedness.	19 (73)
Broomfield and Espie (2003)	RCT	34, primary insomnia, 25 years, 56%	PI versus monitoring control	Sleep diary (SOL, SE, sleep effort), actigraphy (SOL, SE), scale (SAS, SPAQ)	PI > monitoring control: sleep effort, SAS, and SPAQ.	20 (77)
Buchanan (1988)	RCT	33, sleep-onset insomnia, NR, NR	PI versus placebo versus waitlist	Sleep diary (SOL, SE, morning restedness), scale (SPAS)	PI > waitlist at post-treatment and follow-up: SPAS.	20 (77)
Byrne (1985)	Multiple baseline	16, sleep-onset insomnia, 38 years, NR	PI (depressed) versus PI (non-depressed) versus relaxation training (depressed) versus relaxation training (non-depressed) ^a	Sleep diary (SOL, SQ, NAW, SE), sleep monitoring unit (SOL)	PI (non-depressed): reduced subjective and objective SOL. PI (depressed): decreased SQ.	17 (65)
Espie et al., (1989)	RCT	70, sleep-onset insomnia, 45 years, 67%	PI versus relaxation versus stimulus control versus placebo versus no treatment	Sleep diary (SOL, TST, SQ), scale (ZAS, ZDS, SBRS, ARS)	PI: decreased SOL and increased TST and SQ. PI: reduced ZAS, ZDS, and ARS. PI < stimulus control: SOL. PI maintained effect at four follow-ups (the last at 17 months). PI: increased SQ at follow-ups.	20 (77)
Fogle and Dyal (1983)	RCT	33, insomnia, 41 years, NR	PI "Give-up trying" (GUT) versus PI "Try giving-up" (TGU) versus control information	Sleep diary (sleep efficiency, morning restedness), scale (SPA)	Both PI forms = control: sleep efficiency. Both PI forms > control: SPA. No follow-up.	20 (77)
Ladouceur and Gros-Louis (1986)	RCT	27, sleep-onset insomnia, 42 years, 67%	PI versus stimulus control versus sleep information versus monitoring control	Sleep diary (SOL)	PI and stimulus control > sleep information and Monitoring control: SOL. PI = stimulus control at post-treatment and follow-up: SOL.	15 (58)
Ott et al., (1983)	RCT	56, sleep-onset insomnia, 18-55 years, 61%	PI versus PI +feedback versus feedback versus no treatment	Sleep diary (SOL, NAW, SQ), sleep monitoring unit (SOL)	PI: reduced subjective and objective SOL. PI + feedback: increased subjective and objective SOL. Feedback: decreased subjective and objective SOL. No follow-up.	17 (65)
Turner and Ascher (1979)	RCT	50, primary insomnia, 39 years, 50%	PI versus stimulus control versus progressive relaxation versus placebo versus waitlist	Sleep diary (SOL, NAW, restedness, TST, and difficulty falling asleep)	PI > placebo and waitlist: SOL, NAW, restedness, and difficulty falling asleep. PI = stimulus control = progressive relaxation. No follow-up.	19 (73)

ARS, Analogue Rating Scale; NAW, number of awakenings; NR, not reported; PI, paradoxical intention; RCT, randomised controlled trial; SAS, Sleep Anxiety Scale; SE, sleep efficiency; SOL, sleep onset latency; SPA, Sleep Performance Anxiety; SPAQ, Sleep Performance Anxiety Questionnaire; SQ, sleep quality; TST, total sleep time; ZAS, Zung Self-Rating Anxiety Scale; ZDS, Zung Self-Rating Depression Scale.

^aIn the meta-analytical calculations, the two paradoxical intention groups were combined and also so were the two relaxation groups.

TABLE 2 Description of the delivery of paradoxical intention in the included studies

	PI instructions	Administration of PI	Manual	Therapist type, training, and supervision	Treatment integrity check
Ascher and Turner (1979)	(1) Attempt to remain awake as long as possible rather than continuing the effort to fall asleep, (2) lie in bed, in a darkened room, keeping eyes open as long as possible, and (3) not engage in sleep-incompatible activity.	Individual format, 4 sessions (30–45 min each)	NR	NR, NR, NR	NR
Ascher and Turner (1980)	Type A: same as Ascher 1979. Type B: same as Ascher 1979 but with a desensitisation rationale.	Individual format, 4 sessions (30–45 min each)	NR	NR, NR, NR	NR
Broomfield and Espie (2003)	(1) At lights out: stay awake for as long as possible by keeping eyes open, (2) resist sleep-onset gently but persistently in an environment conducive to sleep, and (3) not engage in active methods to stay awake.	Self-help format for 2 weeks	NR	No therapist	No
Buchanan (1988)	Same as Ascher 1979.	Individual format, 3 sessions (20–30 min each)	NR	Psychology students, training in PI	Independent ratings of integrity through video-tape recordings
Byrne (1985)	(1) Remain awake as long as possible each night and pay close attention to whatever thoughts come into your head, (2) try and stay awake with your eyes open, lying as quietly as possible for at least as long as your worst night, if not longer, and (3) in the morning, write down the most common thoughts on the bottom of the sleep log.	Self-help format for 3 weeks	No	No therapist	No
Espie et al., (1989)	Same as Ascher 1979.	Self-help format for 8 weeks	No	No therapist	No
Fogle and Dyal (1983)	Type TGU (try giving-up): (1) Performance anxiety as an explanation for insomnia, and (2) eliminating conscious sleep intention. Type GUT (give-up trying): (1) Anxious sleep effort as triggering night-time tension and morning unrestedness, and (2) removing sleep effort.	Self-help format for 3 weeks	Yes	No therapist	No
Ladouceur and Gros-Louis (1986)	Same as Ascher 1979.	Group format, 4 sessions (120–180 min each)	NR	NR, NR, NR	NR
Ott et al., (1983)	Same as Byrne, 1983.	Self-help format for 2 weeks	No	No therapist	No
Turner and Ascher (1979)	(1) Remain awake at night, and (2) not engage in sleep-incompatible activities.	Individual format, 4 sessions (30–45 min each)	NR	PhD student, NR, NR	NR

NR, not reported; PI, paradoxical intention.

4 | DISCUSSION

4.1 | Summary of main results

The present study is the first comprehensive systematic review and meta-analysis of the effectiveness of PI for insomnia. Relative to passive comparators, PI resulted in large improvements in several central insomnia symptoms. Although the effectiveness of PI

was smaller compared to active comparators, the effects were still moderate for several key outcomes. Relative to previous reviews, the present study extends the quantitative assessment of PI as an evidence-based intervention in that it compared PI with passive versus active comparators and included both night-time and daytime symptoms (Jansson-Fröjmark & Norell-Clarke, 2018; Morin et al., 1999, 2006). A unique finding was support for great reductions in sleep-related performance anxiety by PI. This finding strengthens

TABLE 3 Aggregated effects for the passive comparators

Outcome	Study	Hedge's <i>g</i> (95% CI)	Heterogeneity (95% CI)	Egger's test	Fail-safe N
Sleep onset latency	Ascher and Turner (1979)	-1.48 (-2.56, -0.41)			
	Ascher and Turner (1980)	-0.89 (-1.69, -0.10)			
	Broomfield and Espie (2003)	-0.61 (-1.30, 0.08)			
	Buchanan (1988)	-0.44 (-1.28, -0.41)			
	Espie et al., (1989)	-1.33 (-2.51, -0.51)			
	Ott et al., (1983)	0.00 (-0.64, 0.64)			
	Turner and Ascher (1979)	-1.51 (-2.50, -0.38)			
	Aggregate	-0.82 (-1.25, -0.38)	$I^2 = 49.92\%$ (0.00, 89.39) $Q = 11.99$	-2.85**	64***
Difficulty falling asleep	Ascher and Turner (1979)	2.07 (0.89, 3.25)			
	Ascher and Turner (1980)	1.30 (0.47, 2.12)			
	Turner and Ascher (1979)	2.08 (1.00, 3.17)			
	Aggregate	1.71 (1.12, 2.29)	$I^2 = 3.82\%$ (0.00, 96.63) $Q = 1.77$	$Z = 1.30$	36*
Number of awakenings	Ascher and Turner (1979)	-1.52 (-2.60, -0.44)			
	Ascher and Turner (1980)	-0.82 (-1.60, -0.03)			
	Turner and Ascher (1979)	-1.20 (-2.15, -0.24)			
	Aggregate	-1.10 (-1.63, -0.57)	$I^2 = 0.00\%$ (0.00, 95.33) $Q = 1.11$	$Z = 1.05$	17***
Total sleep time	Ascher and Turner (1980)	0.43 (-0.34, 1.20)			
	Espie et al., (1989)	0.66 (-0.10, 1.42)			
	Turner and Ascher (1979)	0.42 (-0.47, 1.31)			
	Aggregate	0.51 (0.05, 0.97)	$I^2 = 0.00\%$ (0.00, 77.67) $Q = 0.24$	-0.25	3*
Restedness	Ascher and Turner (1979)	2.05 (0.87, 3.22)			
	Ascher and Turner (1980)	1.79 (0.90, 2.67)			
	Buchanan (1988)	0.66 (-0.19, 1.52)			
	Fogle and Dyal (1983)	0.27 (-0.43, 0.98)			
	Turner and Ascher (1979)	1.92 (0.86, 2.99)			
	Aggregate	1.27 (0.53, 2.01)	$I^2 = 69.14\%$ (19.41, 96.26) $Q = 13.47^{**}$	$Z = 3.03^{**}$	60***
Sleep efficiency	Broomfield and Espie (2003)	0.25 (-0.43, 0.92)			
	Buchanan (1988)	-0.27 (-1.11, 0.57)			
	Fogle and Dyal (1983)	0.04 (-0.66, 0.74)			
	Aggregate	0.04 (-0.38, 0.46)	$I^2 = 0.00\%$ (0.00, 94.70) $Q = 0.88$	-0.89	0
Sleep performance	Broomfield and Espie (2003): SAS	-0.96 (-1.67, -0.25)			
	Broomfield and Espie (2003): SPAQ	-1.05 (-1.77, -0.33)			
	Buchanan (1988): SPAS	-1.13 (-2.03, -0.23)			
	Aggregate	-1.04 (-1.48, -0.60)	$I^2 = 0.00\%$ (0.00, 42.23) $Q = 0.09$	-0.24	21***

SAS, Sleep Anxiety Scale; SPAQ, Sleep Performance Anxiety Questionnaire; SPAS, Sleep Performance Anxiety Scale.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 4 Aggregated effects for the active comparators

Outcome	Study	Hedge's <i>g</i> (95% CI)	Heterogeneity (95% CI)	Egger's test	Fail-safe N
Sleep onset latency	Ascher and Turner (1979)	-0.62 (-1.63, 0.38)			
	Ascher and Turner (1980)	-0.22 (-0.98, 0.55)			
	Buchanan (1988)	0.11 (-0.73, 0.94)			
	Byrne (1983)	0.74 (-0.28, 1.75)			
	Espie et al., (1989)	-0.51 (-1.10, 0.09)			
	Ott et al., (1983)	0.63 (-0.02, 1.29)			
	Turner and Ascher (1979)	-0.07 (-0.79, 0.64)			
	Aggregate	-0.00 (-0.39, 0.38)	$I^2 = 41.19\%$ (0.00, 88.24) $Q = 10.28$	0.30	0
Difficulty falling asleep	Ascher and Turner (1979)	1.36 (0.27, 2.45)			
	Ascher and Turner (1980)	0.79 (0.00, 1.57)			
	Turner and Ascher (1979)	0.22 (-0.50, 0.94)			
	Aggregate	0.69 (0.09, 1.29)	$I^2 = 35.15\%$ (0.00, 98.55) $Q = 3.14$	$Z = 1.63$	7**
Number of awakenings	Ascher and Turner (1979)	-1.13 (-2.19, -0.08)			
	Ascher and Turner (1980)	-0.36 (-1.13, 0.40)			
	Byrne (1983)	-0.78 (-1.80, 0.24)			
	Turner and Ascher (1979)	-0.32 (-1.04, 0.40)			
	Aggregate	-0.55 (-0.97, -0.12)	$I^2 = 0.00\%$ (0.00, 90.23) $Q = 1.98$	$Z = -1.357$	7**
Total sleep time	Ascher and Turner (1980)	-0.04 (-0.80, 0.72)			
	Espie et al., (1989)	0.23 (-0.36, 0.82)			
	Turner and Ascher (1979)	0.10 (-0.62, 0.82)			
	Aggregate	0.12 (-0.27, 0.51)	$I^2 = 0.00\%$ (0.00, 83.62) $Q = 0.32$	-0.54	0
Restedness	Ascher and Turner (1979)	1.03 (-0.01, 2.07)			
	Ascher and Turner (1980)	1.12 (0.31, 1.93)			
	Buchanan (1988)	-0.24 (-1.08, 0.60)			
	Turner and Ascher (1979)	0.39 (-0.33, 1.11)			
	Aggregate	0.55 (-0.07, 1.16)	$I^2 = 52.88\%$ (0.00, 96.71) $Q = 6.32$	$Z = 0.54$	6*

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

the notion that decreased performance anxiety is a mechanism through which PI might work.

Cumming and Finch (2001) have recommended that effect sizes should be compared to other relevant estimates in the literature to grasp their significance. In one of the largest and more recent meta-analysis, cognitive and behavioural interventions (e.g. CBT-I, relaxation, stimulus control, psychoeducation, and sleep restriction) were compared with passive comparators (van Straten et al., 2018). Comparing the effect sizes from van Straten et al., (2018) for cognitive and behavioural therapies with the present study's effect sizes for PI relative to passive comparators, the effects were larger in the present study for PI on SOL (0.57 versus 0.82), NAW (0.28 versus 1.10), and TST (0.16 versus 0.51), and smaller on SE (0.71 versus 0.00). Although inferences from comparisons of this sort are difficult to draw from a methodological viewpoint, a reasonable conclusion

would be to state that PI tentatively has a similar effectiveness as other cognitive and behavioural interventions. At the same time, this conclusion is hampered by several limitations in the trials exploring the effectiveness of PI. The relatively few studies, limited number of study participants, and other methodological characteristics of the studies makes an overall conclusion about effectiveness and generalisability of PI uncertain.

4.2 | Methodological considerations and quality of evidence

The present review identified 10 studies that evaluated the effectiveness of PI. There were a number of notable methodological limitations of the studies. The study quality assessment showed that

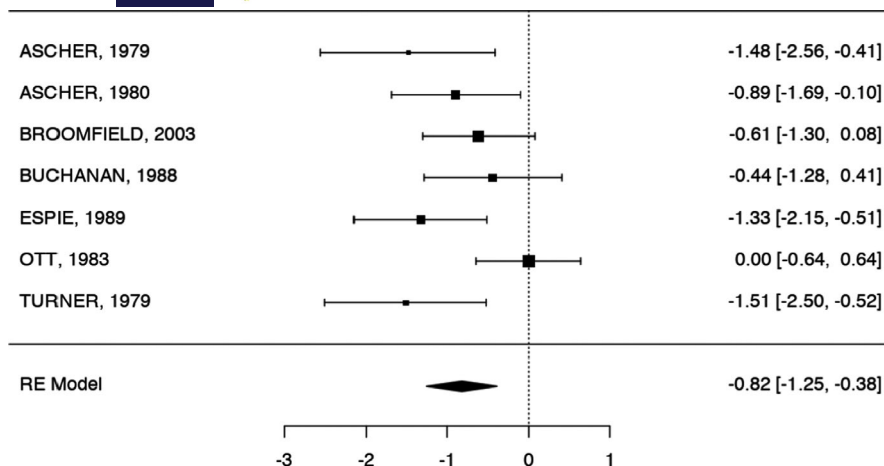


FIGURE 2 Forest plot for sleep onset latency, comparing paradoxical intention to passive comparators

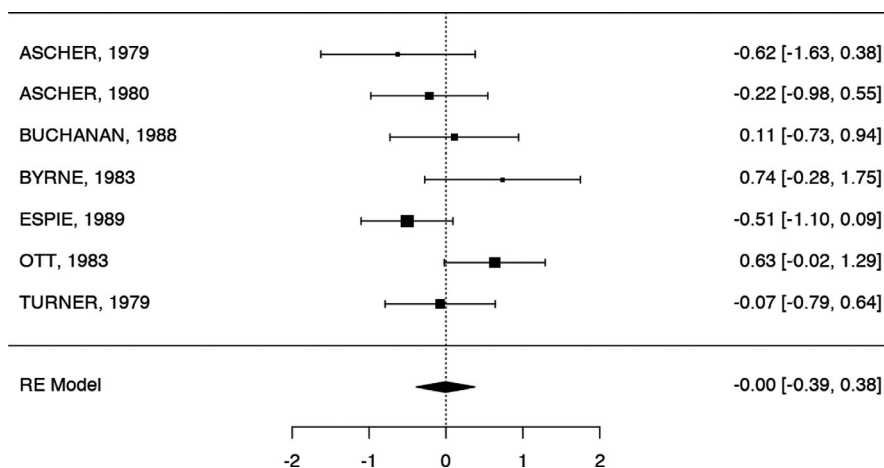


FIGURE 3 Forest plot for sleep onset latency, comparing paradoxical intention to active comparators

the quality of the 10 studies ranged from 15 to 20 points out of 26, implying a moderate study quality. The methodological quality was particularly weak in two areas. First, no studies reported using blinding of subjects, even though it appeared as if this would have been possible. Second, it was uncommon that studies appeared to have sufficient power to detect group differences. While some of these limitations were noted in the study quality assessment, others will be underscored more specifically below.

Across the 10 studies, there was diversity concerning the design. In nine trials, PI was compared with a passive comparator, which means that non-specific factors (e.g. therapist contact) were not controlled for in the estimations comparing PI with passive comparators. Concerning design, it is also worth underscoring that the aggregation of various active comparators into one active comparator category was based on that they provided study participants with active treatment content. This aggregation could, however, have resulted in that comparators with differing effects were combined, so that the comparison between PI and active comparators becomes uncertain.

Another limitation regards the patient characteristics. The total sample size was limited to <400 participants, and none of the trials reported that power calculations were made prior to study start. In all, Type 2 errors are likely, particularly when active treatments were compared. Further, all participants were recruited from the

community, which might make the present findings less generalisable to health settings, as patients in clinical settings tend to display elevated symptoms (Davidson et al., 2009). Another observation is that, in almost all of the studies, we categorised the participants as meeting criteria for sleep-onset insomnia or primary insomnia. Therefore, it is uncertain whether PI should be viewed as an effective intervention for other types of insomnia, such as comorbid insomnia. It is also worth noting that there might be specific insomnia profiles that are particularly susceptible to PI. For example, Espie et al., (2006) have proposed that PI might be specifically suited for patients with psychophysiological insomnia, as this profile of patients are believed to be characterised by attentional bias, pre-occupation with sleep, and using several strategies to avoid sleeplessness. In future research, the study of PI and the effectiveness for different insomnia profiles might also be based on recent empirical attempts to subtype insomnia (Blanken et al., 2019). On a related note, we observed that comorbidity was not formally assessed in the included studies. Although several studies used certain criteria to assess and/or exclude comorbidity, the lack of validated assessments of psychiatric and somatic conditions limits generalisability. As comorbid problems are more common than “pure” insomnia (Stepanski & Rybarczyk, 2006), the lack of assessing comorbid conditions and exclusion of participants with comorbid problems are problematic.

Another issue of methodological uncertainty concerns the administration of PI. There were slight variations concerning several features of the delivery. The rationale and instructions varied across studies, although the original approach by Ascher and Efran (1978) was most commonly employed. Also, the delivery format was mixed, with individual, self-help, and group formats identified. Further, in several treatment-related parameters, it was rare that sufficient information was provided; this concerned whether a treatment manual was used, who delivered PI, whether the therapists were trained and/or supervised, and whether treatment integrity was assessed. Also, the dose of PI varied across studies. Often, PI was delivered across 2–4 weeks, but longer treatment periods were also identified. Based on the limited number of studies in the present review, we were unable to investigate whether certain formats of delivery of PI was more effective than others. During the review process, we also noted that none of the studies assessed treatment-relevant domains that might have importance for the interpretation of findings, such as acceptability, adherence, credibility and expectancy ratings, and perceived usefulness of PI. It should also be emphasised that worsened sleep after PI has been reported in the research literature (Espie & Lindsay, 1985). As none of the included studies in the present review reported on adverse events or deterioration, more research is warranted to examine whether PI produces negative effects among patients with insomnia in general or in subgroups of patients.

An inclusion criterion for the present review was that trials must report insomnia-related outcomes (i.e. night-time and/or daytime symptoms). Across studies, it was less common to index objective sleep outcomes, daytime symptoms, theory-derived processes, and global insomnia symptoms [e.g. with the Insomnia Severity Index; (Bastien et al., 2001)]. Due to the lack of studies assessing several outcome domains, all meta-analytical estimations were based on sleep diary or questionnaire data assessing sleep performance anxiety. As a result, we can only draw conclusions for PI concerning sleep diary-assessed night-time symptoms and, to a lesser extent, sleep performance anxiety. A related limitation is that estimations of effectiveness for PI was not possible to assess in the longer term, as there were not sufficient data for such calculations.

A further limitation is that sensitivity and moderator analyses were not employed due to the limited number of studies. For example, it would have been interesting to explore the effects of the addition or removal of lower quality studies and, to examine whether insomnia symptomatology at baseline and PI administration might moderate the effectiveness of PI. A final limitation is that it was required that the included studies were published in English, thereby introducing a possible language bias.

4.3 | Putative mechanisms

In the present study, we identified three studies that assessed sleep-related performance anxiety as a putative mechanism, and no trial indexing other potential mechanisms (e.g. sleep intention). As a whole, performance anxiety was reduced to a large

degree after PI in the included trials. However, it is important to emphasise that this does not imply that performance anxiety has been demonstrated to act as a putative mechanism. As all trials in the present review analysed sleep-related performance anxiety only as pre- to post-treatment changes, future research might design studies so that mediational analyses become possible. In such studies, repeated assessment of mediators is necessary, and then analysing whether change in mediators precede improvements in insomnia symptoms. This would pave the way for evidence-based explanations for how PI produces improvements (Kazdin, 2007).

Another important methodological aspect of the research literature on performance anxiety is that the self-report scales used in the three studies have not been systematically validated in psychometric terms (Broomfield & Espie, 2003; Buchanan, 1988; Fogle & Dyal, 1983). As a result, it is uncertain whether the construct validity of the self-report scales is sufficiently captured, so that conclusions about sleep performance anxiety can be drawn in the present review. Concerning the measurement of sleep performance anxiety, it should be noted that validated self-report scales are available, such as the Glasgow Sleep Effort Scale (Broomfield & Espie, 2005; Meia-Via et al., 2016; Vand et al., 2020), and such instruments are recommended for future research. The use of validated measures in future trials would enable stronger conclusions about the effectiveness of PI on sleep performance anxiety as well as the possibility to examine mediation in a more rigorous way and explore moderation (e.g. whether PI is particularly effective among insomnia patients with elevated sleep performance anxiety).

One should note that sleep-related performance anxiety is not the only candidate as a putative mechanism for PI. First, PI could be viewed as an intervention that exposes patients to learned, feared stimuli in the bed or bedroom (Lundh, 1998), which enables extinction and the formation of new learning (Craske et al., 2014). However, this notion has not yet been articulated in detail in the research literature and not examined empirically. A second putative mechanistic pathway is described in the attention–intention–effort model (Espie et al., 2006). Although the pathway by Espie et al., (2006) appears to have high face validity, the model has not, to our knowledge, been explicitly tested in its full complexity in the realm of PI treatment.

4.4 | Future directions

There are several important areas that future research could focus on to enhance the understanding of PI. Following from the limitations and uncertainties described above, we recommend future research to use active comparators, sample sizes based on power calculation, samples from clinical settings, a variety of insomnia types (including insomnia disorder), formal assessments of comorbidity, different delivery formats, broad assessments of insomnia symptoms and correlates as outcomes, and different mediators to examine mechanistic pathways.

One unknown dimension of PI is the optimal dosing and administration. Although PI has commonly been implemented by patients during a 2–4-week period, it could be argued that shorter administration of PI could be beneficial as well. Based on the theoretical rationale; that is, breaking a vicious cycle of sleep intention and associated performance anxiety, PI could potentially also be delivered as a behavioural experiment, during which patients test their predictions (e.g. “If I do not try to fall asleep, I will remain awake all night”), followed by testing PI for a limited number of nights. Another topic for future research is the optimal treatment rationale and instructions for PI. Based on two studies included in the present review (Ascher & Turner, 1980; Ott et al., 1983), it appears likely that PI with a desensitisation rationale or with feedback is less beneficial than the original approach by Ascher and Efran (1978). Beyond that, the ideal rationale and instructions remains unknown when delivering PI.

Based on the findings in the present review, the notion of how PI should be used warrants reflection. On the one hand, we believe that CBT-I should still be regarded as the first-line intervention for insomnia disorder (Riemann et al., 2017). On the other hand, PI might play a role in some cases. For example, if a patient remains unimproved after CBT-I, PI could be one option. Also, if the patient reports high sleep-related performance anxiety, and this appears as the primary maintaining factor, PI could be used in isolation or in combination with other efficacious CBT-I components, such as sleep restriction (Miller et al., 2014). To date, current CBT manuals do not include PI as a treatment component (van Straten et al., 2018). Whether the addition of PI could add efficacy to CBT-I is currently unknown. Future research could explore the notion of combining PI with CBT-I to explore potential additive effects, but also whether there are subgroups of patients who benefit more from PI.

5 | CONCLUSIONS

Although the research literature is limited and some methodological uncertainties remain, we conclude that PI is an effective intervention for insomnia, particularly so for reducing sleep initiation and maintenance difficulties, as well as increasing the perception of feeling rested after sleep. The effectiveness and implementation of PI need to be examined further, both as a stand-alone intervention and within the realm of CBT-I.

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CONFLICTS OF INTEREST

None of the authors have any conflicts of interest to declare.

AUTHOR CONTRIBUTION


MJF and ANC conceived and designed the meta-analysis. SA, BB, and AR extracted and analysed the data. MJF wrote the paper with

important contributions from SA, BB, AR, and ANC. All authors participated in the review and revision of the manuscript and have approved the final manuscript to be published.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Markus Jansson-Fröjmark  <https://orcid.org/0000-0003-2059-1621>

Annika Norell-Clarke  <https://orcid.org/0000-0003-2008-0784>

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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