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The Effect of Interpersonal Psychotherapy for Depression on Insomnia Symptoms in a Cohort of Women with Sexual Abuse Histories

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Abstract

Insomnia frequently occurs with trauma exposure and depression, but can ameliorate with improvements in depression. Insomnia was assessed by the insomnia subscale of the Hamilton Rating Scale for Depression in 106 women with childhood sexual abuse (CSA) and major depression receiving Interpersonal Psychotherapy in an uncontrolled pilot (n = 36) and an immediately subsequent randomized controlled trial (n = 70) comparing IPT to treatment as usual. Depression improved in each study and in both treatment conditions; insomnia had smaller, non-significant improvements. Overall, 95 women (90%) endorsed insomnia on the Structured Clinical Interview for DSM-IV at baseline and, of those, 90% endorsed insomnia following treatment. Despite improvements in depression, insomnia persists for most women with CSA.

Trauma related to childhood sexual abuse (CSA) is associated with psychiatric and interpersonal difficulties during adulthood, including depression and anxiety (Kendler et al., 2000). Less attention has been focused on insomnia, the difficulty of initiating and maintaining sleep, in the context of CSA. In general, insomnia is strongly linked to depression (Pigeon & Perlis, 2007) with approximately 90% of individuals with major depressive disorder (MDD) also experiencing sleep disturbances (Riemann & Voderholzer, 2003).

Insomnia symptoms tend to persist following various treatments for MDD including medication treatment (Menza, Marin, & Opper, 2003), cognitive behavior therapy (CBT; Carney, Segal, Edinger, & Krystal, 2007; Manber, et al., 2003), and stepped care depression management (Pigeon et al., 2008). This evidence suggests that insomnia may not reliably improve with these forms of depression treatment.

Interpersonal psychotherapy (IPT) has recently been evaluated for the treatment of depression in CSA survivors, where it was found to decrease depression in an uncontrolled pilot study (Talbot et al., 2005) as well as in a subsequent randomized trial (RCT) comparing IPT to treatment as usual (TAU) (Talbot, Ward & Lu, 2008). The current study undertook secondary analyses of both sets of clinical trial data to specifically evaluate the effect of IPT on insomnia. The hypotheses were that there would be a significant effect of IPT treatment over time for insomnia, that insomnia would improve more with IPT than

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with TAU in the RCT, and that the effect sizes would be relatively smaller for insomnia than for depression.

Method

Participants

The sample is derived from two closely related studies in depressed women with CSA presenting at a community mental health center. During intake assessments, 1,080 women were screened for eligibility, consisting of depressive symptoms and self-report of CSA. Of 163 eligible women, 133 agreed to be contacted for evaluation, of which 22 were excluded (8 with alcohol/substance abuse, 6 with active psychosis, 6 without MDD, and 2 without CSA), 4 did not complete the baseline evaluation, and 1 withdrew immediately. Thus, 106 participants with a history of CSA established through a structured interview of CSA (Talbot et al., 1999) and MDD established through the Structured Clinical Interview for DSM-IV (SCID-I; Spitzer, Gibbon, & Williams, 1994) were enrolled. Of these, 36 enrolled in an uncontrolled pilot study of IPT; an additional 70 were enrolled in an immediately subsequent RCT and were randomized to IPT (n = 37) or TAU (n = 33).

Overall participants in the combined studies (N = 106) had an average age of 36 (SD = 9) years and were ethnically diverse (50% African American or Hispanic), economically disadvantaged (38% single mothers and 61% unemployed), and mental health burdened (68% had ≥ 3 Axis I diagnoses). Pilot study participants were approximately 4 years younger than those in the RCT and more often African American or Hispanic (67% compared to 42%). The two RCT treatment groups (n = 70), which are directly compared in some analyses, did not differ with respect to severity of depression, psychiatric burden, or baseline demographic variables with the exception of age (see Table 1).

Measures

Baseline assessment included the SCID-I, both to determine eligibility and assess comorbid Axis I disorders, while the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and Beck Depression Inventory-II (BDI-II; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) were used to assess depression severity. At 10, 24 and 36 weeks after enrollment, BDI-II, and HRSD were re-administered to assess depression status as the primary outcome; the SCID current major depression module was re-administered at 24 and 36 weeks. A single assessor was used who was not blinded to participants' group assignments in the RCT.

For the current analysis, three sleep items from the HRSD (early, middle, and late insomnia) were summed to arrive at an insomnia score. These items have been observed to load on the same HRSD factor (Fleck et al., 1995), to detect improvement in insomnia associated with effective treatment of depression (Thase et al., 2002), and to have convergent validity with prospective measures of insomnia derived from a week of daily sleep diaries (Manber et al., 2005). For all depression analyses, sleep items were removed from both the HRSD (3 items) and the BDI-II (2 items).

Procedure

Manualized IPT was administered for 14 weekly, individual sessions followed by 2 biweekly sessions and monthly sessions as clinically indicated by 6 master's level and 3 doctorate level clinicians at a community mental health center. Treatment focused on current interpersonal difficulties including grief or loss, interpersonal disputes, role transitions, and the impact of past abuse on present interpersonal functioning. Training in IPT consisted of a two-day seminar, 15 hours of didactics in weekly group supervision, and completion of 3

audio-taped or video-taped training cases that met trainer criteria for adherence and competence in IPT delivery. Therapist fidelity to the structured therapy was monitored in weekly group supervision using taped sessions for the duration of the studies. Women receiving IPT in either the pilot or RCT completed $M = 12.0$, $SD = 6.8$ sessions with 77% receiving ≥ 6 sessions, which was found to be a therapeutic dose of psychotherapy for depression in a similar sample (Miranda et al, 2003).

TAU was typically delivered in bi-weekly individual sessions by 4 master's level staff therapists in the same setting using a variety of standard interventions including supportive (53%), cognitive-behavioral or dialectical-behavioral (27%), integrated/eclectic (13%), and client-centered (7%) approaches. Women in the TAU condition completed an average of $M = 6.3$, $SD = 4.2$ sessions with 58% receiving ≥ 6 sessions. The TAU condition, therefore, was an active intervention, but fewer participants received a ≥ 6-session dose of treatment than in the IPT condition.

Data Analysis

An intent-to-treat approach was employed, such that all participants enrolled in the studies were included in analyses. Missing data occurred in 10 of the pilot participants (28%), 6 of the RCT IPT participants (16%), and 11 of the RCT TAU participants (33%). Logistic regressions used to test for a missing pattern did not identify any covariate that predicted the missing data. Accordingly, an assumption was made that the missing pattern was Missing Completely At Random.

To test for a time effect within each treatment condition (pilot IPT, RCT IPT, and RCT TAU) on each of the three primary outcome variables (HRSD without sleep items, BDI-II without sleep items, and insomnia), scores across the four time points (baseline, week 10, week 24, week 36) were assessed using the generalized estimating equations in regression analyses with exchangeable working correlations to address the correlated responses in the data. This approach is widely used due to its less stringent distributional assumptions and robustness properties, yielding valid inference regardless of the data distribution (Liang & Zeger, 1986). All analyses include age as a continuous covariate along with the following dichotomous covariates recorded at baseline: race, posttraumatic stress disorder (PTSD), borderline personality disorder, and antidepressant use. For the insomnia analyses, depression was controlled by adding the change in HRSD (minus sleep items) from baseline to week 36 as an additional covariate.

To compare the IPT and TAU groups in the RCT, the same approach was undertaken adding group as the between-groups contrast. Hedge's g, which adjusts for small sample sizes (Hedges, 1981), was calculated to determine across-treatment effects over time and to determine the effect size of IPT relative to TAU in the RCT (between-group effect). Pearson's r were calculated for change scores on the depression scales and the insomnia change score to determine to what extent changes in depression and insomnia were related.

An assessment of a possible dose-response relationship was first undertaken by calculating Spearman's rank-order correlations between the number of treatment sessions received (dose) and pre-post change scores (treatment response) for each outcome variable in each condition. In addition, the effect of receiving a minimal therapeutic dose of treatment was assessed by creating a dichotomous variable (≥ 6 sessions or < 6 sessions) and adding this variable to models in subsequent analyses to determine whether it altered between-group effects in the RCT.

Results

Depression scores absent sleep items on both the HRSD and the BDI-II significantly improved across time in the uncontrolled IPT group (both $p < .001$), where the effect sizes for IPT were $g = 0.92$ and $g = 1.32$, respectively. No significant improvements are noted for insomnia, where the effect size was $g = 0.49$. Results are further detailed in Table 2.

HRSD and the BDI-II depression scores absent sleep items significantly improved in the RCT IPT group with $\chi^2(3, N = 36) = 17.56$, $p < .001$ and $\chi^2(3, N = 36) = 13.94$, $p < .001$, and effect sizes of $g = 1.10$ and $g = 0.85$, respectively. Depression scores, however, also improved in the TAU condition, albeit these improvements were more modest (see Table 2). Across-treatment effect sizes for TAU were $g = 0.76$ for the HRSD and $g = 0.64$ for the BDI-II. In contrast, after controlling for improvement on the HRSD as an additional covariate, insomnia scores did not significantly improve in either the IPT or TAU condition with $\chi^2(3, N = 36) = 6.94$, *ns* and $\chi^2(3, N = 32) = 4.60$, *ns*, although a trend was evident at the level of $p = .07$ in the IPT condition. Across-treatment effect sizes for insomnia were smaller than those observed for depression

In group x time contrasts, participants in the IPT condition improved relative to the TAU condition on the HRSD with $\chi^2(3, N = 68) = 5.00$, $p < .05$ and BDI-II with χ^2 $(3, N = 68) = 4.25$, $p < .05$. Effect sizes for IPT compared to TAU on depression were small to moderate (Table 2). There was a trend for greater improvement in insomnia in the IPT condition with $\chi^2(3, N = 68) = 3.00$, $p = .07$). Effect sizes were small in these between-treatment contrasts for all three variables (Table 2).

No significant relationship was observed between number of treatment sessions and the magnitude of improvement on the HRSD, BDI-II or the insomnia score. Adding the covariate of completing ≥ 6 treatment sessions to the RCT between-group analyses did slightly alter the size of observed group differences from the primary analyses. The IPT condition continued to have greater improvements than the TAU condition on the HRSD (p <.05), while findings on the BDI-II moved from significance to a strong trend ($p = .054$) and insomnia findings moved from a trend to significance ($p < .05$); all effect sizes remained small with $g = 0.29$, 0.26 and 0.30, respectively.

In the overall sample, there was a small but significant correlation between pre-post changes in both the HRSD and the BDI-II (with sleep items removed) and changes in the insomnia scores, $r = .25$, $p < .05$ and $r = .21$, $p < .05$, respectively.

At baseline 95 of the 106 participants (90%) met SCID-I criteria for insomnia, while 85 of those continued to endorse insomnia following treatment (80% of the sample and 90% of those with baseline insomnia). Similarly, 89 participants (84% of the sample) endorsed at least one of the HRSD sleep items as severe at baseline, while 62 of those continued to endorse insomnia following treatment (58% of the sample and 70% of those with severe insomnia). In terms of insomnia subtype, 51% had severe sleep initiation insomnia, 57% had severe middle of the night insomnia, and 30% had severe complaints of early morning awakening (some individuals reported more than one type of severe insomnia); the distribution of these subtypes did not change appreciably following treatment.

Discussion

This secondary analysis assessed the treatment effects of IPT on insomnia in an outpatient sample of women with MDD and histories of CSA. For both the IPT pilot and the RCT, there were significant improvements on depression following IPT, while smaller but significant improvements were also achieved in the TAU condition. Improvements in

insomnia did not reach significance. Comparing IPT to TAU on depression, significant between-group effects favored IPT with a non-significant trend for insomnia to improve.

Although the RCT allows a direct comparison of IPT to TAU, we believe the most interesting findings are the across-treatment effects for each individual treatment on the depression and insomnia measure in both the RCT and the uncontrolled pilot study. Although across-treatment effects tend to be inflated relative to between-group effects, large effects for IPT and a moderate effects for TAU on depression were evident, with more modest effects for insomnia symptoms. These findings were corroborated at the descriptive level. Namely, a large majority of participants presenting with SCID-assessed insomnia or severe HRSD insomnia continued to endorse insomnia following treatment.

The main finding of this study was that IPT for depression in women with CSA histories improved insomnia modestly, but not significantly, after controlling for a number of variables including changes in depression. For the majority of participants, insomnia persisted after IPT treatment. This compares to Zayfert & Deviva's report (2004) that 48% of patients who no longer met criteria for PTSD following exposure therapy for PTSD had residual insomnia. Given that IPT does not include any sleep-specific intervention and that its effect on insomnia was small when compared to TAU, any observed improvements in insomnia may be a random occurrence or related to nonspecific effects present in any form of treatment.

Limitations of the study include the absence of polysomnographic measures of sleep, the absence of longer term outcomes, and that insomnia was assessed by a three item subscale from the HRSD as opposed to a more comprehensive insomnia instrument. However, Manber and colleagues (2005) validated this HRSD subscale against the standard of assessing insomnia by prospective daily sleep diaries. Comorbid PTSD could be associated with insomnia symptom change, but these data were not available. The single assessor was not blind to treatment condition. Participants in the RCT's IPT condition received more treatment sessions, but secondary dose-response analyses did not yield substantially different results than the primary analyses.

The present results are consistent with a growing body of literature suggesting that insomnia symptoms accompanying psychiatric disorders may not fully ameliorate in consort with the primary disorder. This is especially germane since insomnia is associated with chronic illness, lower quality of life, increased probability of relapse and recurrence of MDD, and increased suicide risk (Menza et al., 2003). For CSA survivors in particular, the sleep environment may be uniquely associated with insomnia if the sexual trauma occurred in a sleep context. Consequently, hyperarousal and hypervigilance may become a conditioned response to the bedroom, and sleep may be perceived as dangerous because it restricts the ability to monitor one's surroundings (Craske & Tsao, 2005). These trauma-specific manifestations of insomnia along with the high prevalence rates of insomnia in traumatized populations make apparent the need for a sleep assessment in patients presenting with trauma histories.

When insomnia persists following conventional treatments for MDD and/or PTSD, a targeted insomnia intervention may be required. Preliminary evidence has shown CBT for insomnia (CBT-I) to be effective in alleviating insomnia among patients with PTSD (DeViva, Zayfert, Pigeon, & Mellman, 2005; Germain, Shear, Hall, & Buysse, 2007). Additional research is needed to evaluate the efficacy of CBT-I and/or pharmacotherapy for treating insomnia in individuals with trauma histories, whether insomnia interventions may be delivered in conjunction with therapies such as IPT, and whether any patient-specific factors predict the occurrence of residual insomnia.

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Table 1

Sample Characteristics for the Randomized Controlled Trial

	IPT (*n* = 37)			TAU (*n* = 33)			
Baseline Variable	***M***	***SD***	**%**	***M***	***SD***	**%**	***t* or χ^2 value[a]**
Age	38.6	10.6		33.9	8.4		2.91 *
White			62			54	0.42
Employed			35			27	0.50
Single Parent			40			36	0.02
Total Axis I Diagnoses	3.2	1.1		3.2	1.5		0.01
BDI-II II Total Score	34.4	10.4		34.8	10.3		0.09
HRSD Total Score	26.0	5.8		25.8	5.3		0.17
Insomnia			87			88	0.09
Dysthymia			51			36	1.59
PTSD			62			49	1.84
Panic Disorder			46			61	1.50
Obsessive Compulsive Disorder			27			27	0.01
Hx of Alc/Substance Dependence			46			33	1.16

Note. Diagnoses are from the Structured Clinical Interview for DSM-IV Axis I Disorders. IPT = Interpersonal Therapy; TAU = Treatment as Usual; BDI-II = Beck Depression Inventory; HRSD = Hamilton Rating Scale for Depression.

[a] *t* statistic is reported for comparison of group means, while the χ^2 value is provided for contingency analyses in which all degrees of freedom = 1.

* $p < .05$, two-tailed.

Table 2

Effect of Treatment on Depression and Insomnia.

Variable		Pre[a]		Post[a]		Time[a]		Time x Group[a]	
Group	***n***	***M***	***SD***	***M***	***SD***	**Wald χ^2**	***g***	**Wald χ^2**	***g***
Uncontrolled IPT Pilot Study									
HRSD	36	18.2	5.3	10.5	6.5	19.12***	1.32		
BDI II	36	28.3	10.5	17.4	13.6	19.34***	0.92		
Insomnia	36	2.5	1.5	1.7	1.8	0.69	0.49		
Randomized Controlled Trial									
HRSD									
IPT	*37*	19.3	4.1	13.3	6.6	17.56***	1.10	5.00*	0.29
TAU	*33*	19.6	3.5	15.8	6.5	11.60**	0.76		
BDI II									
IPT	37	30.6	9.5	20.4	14.1	13.94**	0.85	4.25*	0.30
TAU	33	31.1	9.2	24.1	12.7	8.64*	0.64		
Insomnia									
IPT	37	3.8	1.8	2.3	1.7	6.94[b]	0.84	3.30[b]	0.22
TAU	33	3.3	1.7	2.7	1.3	4.60	0.32		

Note. g = Effect size reported as Hedges *g*; HRSD = Hamilton Rating Scale for Depression with sleep items removed; BDI-II = Beck Depression Inventory version 2 with sleep items removed; IPT = Interpersonal Therapy; TAU = Treatment as Usual.

[a]Reported means represent estimated marginal means; Covariates include age, race, and presence/absence of PTSD, borderline personality disorder and antidepressant use at baseline with change in HRSD (absent sleep items) used as additional covariate for insomnia analyses. Time represents the effect of treatment over four time points (only pre and post values reported); effect size based on pre-post change in each condition; Time x Group represents the effect of group over time; effect size based on group differences at the final time point.

[b]A trend was present at $p = .07$ for the IPT time effect and $p = .07$ for the group effect.

*$p < .05$.

**$p < .01$.

***$p < .001$. (two-tailed.)