

A Retrospective Chart Review of Adult Insomnia and Headaches in an Australian Outpatient
Psychology Training Clinic: Prevalence and Effects on Psychological Treatment Response

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Abstract

Objective: Sleep dysfunction and headaches are common and disabling conditions which are frequently comorbid with psychiatric disorders. Furthermore, there is a growing body of literature linking sleep dysfunction to the frequency and intensity of headaches. This study retrospectively examined the prevalence of insomnia and headache frequency, and their effects on general psychological treatment outcomes in a sample of patients attending a university psychology outpatient clinic in metropolitan Queensland, Australia. **Method:** Outcome Questionnaire 45 (OQ45) data were extracted for 69 adult patients who presented to the Griffith University Psychology Clinic (Mount Gravatt, Queensland) in 2018, and whom consented to the use of their de-identified data for research purposes. **Results:** The prevalence of frequent insomnia and headaches in the week of patients' initial session were 47.8% and 23.2%, respectively. Additionally, there was a correlation, $r_s = .26, p = .02$ between frequency of insomnia and headaches at the initial session. Frequent insomnia (Odds Ratio = 4.62, $p < .01$), headaches (OR = 3.82, $p = .055, p_{bootstrapped} = .04$), and comorbid insomnia and headaches (OR = 7.25, $p = .02$) were strong predictors of inadequate treatment response, adjusting for age, sex, and number of therapy sessions. Effect sizes achieved by students were large ($d = 0.9$), and similar to those found in studies of fully qualified therapists. **Conclusions:** Our study demonstrates clinicians should be aware that sleep and headache problems may complicate psychological treatment of other problems, and that presence of these factors should warrant specific targeted attention early in treatment.

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Significant sleep problems (such as insomnia, sleep apnoea, and restless legs syndrome) are known to affect approximately 40% of the Australian population (Adams, Appleton, Taylor, McEvoy, & Antic, 2016), and it is estimated that 20.55% experience migraine headaches (Deloitte Access Economics, 2018). There is an growing literature demonstrating the link between sleep problems and headache (Barloese, Jennum, Knudsen, & Jensen, 2012; Barloese, Lund, & Jensen, 2014; Rains & Poceta, 2006), including an Australian study by Sullivan and Martin (2017) which found relationships between increased headache activity and inadequate sleep quality and duration, circadian rhythms, and sleep apnoea risk in a non-clinical sample. The comorbidity of sleep problems and headaches are frequent. A total of 66% of female chronic migraineurs in Calhoun, Ford, Finkel, Kahn, and Mann (2006) reported sleep-onset insomnia, and over 80% of the sample reported waking unrefreshed. Similarly, over half of the sample of migraineurs in Kelman and Rains (2005) reported frequent difficulties with sleep initiation or maintenance. From a patient's perspective, a meta-analysis of studies examining perceived headache triggers found sleep problems to be the second most commonly reported trigger by headache patients (Pellegrino, Davis-Martin, Houle, Turner, & Smitherman, 2018). The comorbidities between sleep and headaches likely relate to shared neurological structures between sleep and pain, such as the hypothalamus (Brennan & Charles, 2009). A recent study also implicated neuroendocrine mechanisms, with insomnia being shown to be a strong moderator in the relationship between the inflammatory marker, C-reactive protein, and migraine (Hagen, Hopstock, Elise Eggen, Mathiesen, & Nilsen, 2019). Additionally, headache coping behaviours such as napping or certain medication choices may further dysregulate sleep and its rhythms, leading to a perpetuation of a chronic headache disorder (Ong & Park, 2012). To date, the relationships

between sleep and headache problems have not been examined in an Australian clinical psychology sample. More broadly, there is a paucity of research on the prevalence of sleep problems, headaches, and their comorbidity in clinical psychology outpatient settings. One study from the University of New South Wales found the 6-year prevalence rate (1989-1995) of sleep disorder presentations in their clinic for postgraduate clinical psychology trainees to be 7.1%, 4.3% of which were insomnia (Murrell, Steel, Gaston, & Proudfoot, 2002). Unfortunately, there do not appear to be studies reporting on the prevalence of sleep disorders, or headaches in psychological practice in more contemporary samples than Murrell's.

Whilst headache and sleep disorders are disabling and distressing in their own right, a concern for treating psychologists more broadly, is that they may complicate treatment of other presenting psychopathologies, possibly leading to poor treatment response. For example, Gillin (1998) found sleep disturbance to be a risk factor for mood, anxiety, and substance misuse disorders; and Breslau, Merikangas, and Bowden (1994) report the relationship between headaches and mood disorders, including depression and bipolar affective disorder. Conversely, evidence suggests that treating sleep may improve psychiatric comorbidities; for example, a study by Cunningham and Shapiro (2018) found Cognitive-Behaviour Therapy for Insomnia (CBT-i) may be effective in depressive presentations with a sleep comorbidity. As for the treatment of comorbid sleep and headache presentations, Sullivan, Martin, and Boschen (2019) conducted a meta-analysis showing psychological sleep interventions (including behavioural and cognitive-behavioural techniques) were effective at reducing the frequency of headaches. However, despite the importance of recognising and treating sleep problems in psychological practice, training in sleep disorders and headaches has traditionally not been formally included in postgraduate clinical psychology programs (Meltzer, Phillips, & Mindell, 2009). It has been demonstrated that

sleep education modules for clinical psychology trainees increases their appreciation of sleep's role in physical and mental health, and their ability and confidence to assess sleep problems in their practice (Peachey & Zelman, 2012). As such, inclusion of sleep modules in clinical psychology training programs may help future clinicians recognise and effectively treat these important comorbidities.

There has been no research published examining if Australian postgraduate clinical psychology trainees adequately manage headache problems, and only one study using data from 1989 – 1995 (Murrell et al., 2002) which evaluates Australian student therapists' effects on sleep. Given the gaps in the literature identified, this study addresses the following research questions:

1. What is the prevalence of frequent insomnia, frequent headaches, and comorbid frequent insomnia and headaches at the initial session in adults attending our psychology clinic?
2. In our clinic sample, how strong are the correlations between insomnia and headaches at treatment onset, and how do these compare to relationships seen in non-clinical samples?
3. Are frequent insomnia, frequent headaches, and comorbid frequent insomnia and headaches associated with:
 - a. higher levels of distress at treatment onset?
 - b. poorer treatment response?
4. Do the treatments provided by our sample of postgraduate clinical psychology trainees effectively reduce insomnia and headache symptomatology, and psychological distress more broadly? Furthermore, how do treatment outcomes compare to therapy in non-student settings?

Our study is the first examining the prevalence of insomnia and headaches in an Australian psychology outpatient clinic; as such, hypotheses regarding RQ1 are tentative. Given the prevalence of insomnia in the Australian general population is approximately 20% (Adams et al., 2016), we hypothesise that the prevalence in an outpatient clinical setting will be at least 20% at treatment initiation, likely higher due to an increased level of psychological distress. There is a paucity of peer-reviewed epidemiological data on headache in Australia, however a recent study by Deloitte estimates the prevalence of migraine in Australia to be 20.55% (Deloitte Access Economics, 2018). The 20.55% statistic does not include other common headache presentations such as tension-type headache. As such we hypothesise that the prevalence of frequent headaches in our clinic at initial session would be at least 20.55%, likely higher given the psychiatric comorbidities such as stress, sleep problems, anxiety, and depression are common headache triggers (Pellegrino et al., 2018).

For RQ2, it is hypothesised that the correlation effect size would be $r_s = .3$ or higher, based on an Australian non-clinical sample in Sullivan and Martin (2017) where the correlation between poor sleep quality and migraines was $r_s = .33$, and tension-type headache $r_s = .28$.

As per the findings of Gillin (1998) associating sleep disturbances to a range of psychopathology, and similarly for headaches (Lake III, Rains, Penzien, & Lipchik, 2005), it is expected that frequent sleep and headache problems will be associated with greater psychological distress at treatment initiation and poorer treatment outcomes at termination (RQ3).

In a study of practicing outpatient mental health clinicians, Kraus, Castonguay, Boswell, Nordberg, and Hayes (2011) found small to large therapy effect sizes depending on the presenting issue. Whilst being a topic of some debate, the effect of therapist experience (i.e., years of practice) tends not to account for a substantial part of the variance in therapy

outcomes (Goldberg et al., 2016). As such, we predicted our clinical psychology trainees to achieve at least a moderate effect size for insomnia, headaches, and the OQ-45 total, similar to that of clinicians practicing in the community (Kraus et al., 2011) (RQ4). The prediction for improvement in insomnia and headaches is based on an assumption that even where sleep and headache problems aren't targeted specifically, treatment of the primary presenting concern (regardless of issue) may provide some degree of relief. This may be due to reductions in psychophysiological somatising of distress, and additionally, providing general coping strategies which could be applied to insomnia and headaches (such as relaxation training, restructuring of catastrophizing cognitions etc.)

Method

Participants

Data were extracted from the files of 69 adult therapy patients whom attended our clinic in 2018. Inclusion criteria were that the patient was an adult (aged > 18 years), was presenting for individual therapy, and that they had consented to their de-identified data to be used for research purposes. Cognitive assessment, couple's therapy, and child therapy cases were excluded. Data were not extracted for 54 patients for reasons including the patient not consenting to the use of their data for research ($N = 25$), missing or un-witnessed data consent forms ($N = 3$), failing to attend any appointments ($N = 20$), or database errors whereby no 2018 therapy OQ data were available ($N = 6$). Table 2 presents the characteristics of both the patients and therapists in the sample. The therapists who staffed our outpatient student psychotherapy clinic in 2018 were completing their first clinical year of a Master of Clinical Psychology or Doctor of Philosophy in Clinical Psychology degree at Griffith University, Mount Gravatt Campus. Therapists complete an approximately nine-month period in the University clinic at this stage of training, accruing at least 160 hours of direct patient contact performing adult and child therapy, cognitive assessments, and group therapy. Therapists in

their internship year are trained in Cognitive Behaviour Therapy, however they may occasionally practice other schools of psychotherapy depending on their clinical supervisor's theoretical orientation and recommendations for a particular patient's presentation. As this study was retrospective chart review where consent to examine the files had already been obtained, our study was deemed by our institution's Human Research Ethics Committee to be exempt from ethical review. Extraction, storage, analysis and reporting of patient data was conducted in accordance with the Australian *National Statement on Ethical Conduct in Human Research (2007)*.

Measures

The Outcome Questionnaire-45 (OQ45) (Lambert, Gregersen, & Burlingame, 2004) measures psychiatric symptom distress, interpersonal relations, and social role problems. The OQ45.2 version instrument is administered at every session to therapy patients in our clinic to track treatment progress, reliable clinical change, and to predict treatment prognosis. Items on the OQ45.2 are presented on a 5-point Likert scale: "Never", "Rarely", "Sometimes", "Frequently", "Always/Almost Always." In a setting similar to our clinic, the OQ-45.2 was determined to be reliable and valid; Cronbach's α for subscales and the OQ45.2 total were: symptom distress (.93), interpersonal relations (.78), social role (.7), OQ45.2 total (.94) (Boswell, White, Sims, Harrist, & Romans, 2013). Validity was demonstrated via significant correlations between a range of psychiatric and social/interpersonal presentations (Boswell et al., 2013).

Procedure

For consenting patients, the coder (DS) extracted year of birth and sex, and the following OQ variables from initial and final sessions onto a standardised form: item 41 (insomnia) "I have difficulty falling asleep or staying asleep", item 45 (headaches) "I have headaches", symptom distress subscale, social role subscale, interpersonal relations subscale,

and OQ-45 total. There was a small proportion of missing data whereby items 41 or 45 were skipped by the patient, or where they only attended 1 session. Items related to comorbidities such as mood, and anxiety were not extracted from the OQ-45 based on previous research by Sullivan and Martin (2017), which found depression, anxiety, and stress did not moderate the relationship between sleep quality and migraine and non-migraine headaches.

Statistical Analyses

Frequency and descriptive statistics were used to answer questions relating to the prevalence of insomnia and headache problems in the sample. Where correlations were performed between individual OQ-45 items and the symptom distress and OQ-45 total scales, adjustments were performed to subtract the individual item from the total score to prevent collinearity. For example, when performing a correlation of insomnia with the OQ-45 total, the OQ-45 total minus the value of insomnia was used. For correlations including single OQ-45 items (ordinal data), Spearman's rank order correlations (r_s) were performed and one-tailed significance cut-offs were used as multi-directional effects were not predicted (e.g., greater insomnia frequency would not be expected to be associated with fewer interpersonal relationship problems). To answer the research question of treatment efficacy of sleep and headache problems, frequency statistics, t -Tests and Wilcoxon signed rank tests were calculated to determine the proportion of those with frequent insomnia, and frequent headaches whom no longer reported these issues to be frequent at treatment conclusion. Additionally, Odds Ratios and Chi-Square statistics were calculated to determine the odds of continuing to have sleep or headache problems "frequently" or more at treatment conclusion, when these issues were frequent at treatment onset. Number needed to treat statistics were calculated based on treatment onset and termination prevalence rates of insomnia and headaches. An inadequate treatment response was defined as having an OQ-45 total > 63 at

treatment conclusion, which is the OQ cut-off for clinically significant distress (Boswell et al., 2013).

Power analysis.

Power analyses were conducted *a priori* to determine the number of patients required to find a significant medium effect size, with .8 power at an α level of .05. A Pearson product moment correlation (statistically similar to Spearman's Rho) with a correlation effect size of $r = .3$ required 60 patients. For a 2×2 contingency table χ^2 statistic, 88 patients were required. For a matched pairs *t*-test (one-tailed) assuming a moderate effect size, 27 patients were required.

Results

The one-year-prevalence of frequent insomnia, frequent headaches, and comorbid frequent insomnia and headaches at treatment onset in adults in our clinic was 47.8% (insomnia), 23.2% (headaches), and 15.9% (comorbid insomnia and headaches).

Spearman correlations were performed between insomnia and headache frequency at treatment onset, finding a small significant correlation, $r_s = .26, p = .02$ (one-tailed). Furthermore, for initial session data, correlations were found between insomnia frequency and general psychiatric symptom distress, $r_s = .26, p = .02$, and the OQ-45 total, $r_s = .23, p = .03$. Initial session headache frequency was significantly correlated with psychiatric symptom distress, $r_s = .25, p = .02$, interpersonal relational problems, $r_s = .23, p = .03$, and the OQ-45 total, $r_s = .27, p = .01$, but not the social role subscale. One-tailed tests were used, as a positive directional effect was hypothesised based on previous research linking sleep problems to headaches (Brennan & Charles, 2009; Ong & Park, 2012; Sullivan & Martin, 2017).

Table 1 reports the correlation between insomnia and headache frequency at the initial session and OQ45 subscales at the final session. 2×2 contingency tables were compiled to

compute the χ^2 and Odds Ratio for the association between frequent insomnia, frequent headaches, and comorbid frequent insomnia and headaches at treatment onset, and the presence of clinically significant distress at treatment termination. Frequent insomnia at treatment onset more than quadrupled the odds of being in clinically significant distress at therapy termination, $OR = 5$, $\chi^2 = 9.89$, $p < .01$ (Figure 1). Frequent headaches were similar in magnitude to insomnia in predicting inadequate treatment response, $OR = 4.95$, $\chi^2 = 6.86$, $p < .01$ (Figure 2). Comorbid frequent insomnia and headaches were the strongest predictor of an inadequate treatment response, $OR = 6.85$, $\chi^2 = 6.61$, $p = .01$ (Figure 3).

Table 3 presents regression coefficients for two logistic regression models predictive of inadequate treatment response. Whilst adjusting for age, sex, and number of sessions, a model testing frequent insomnia or frequent headaches as a predictor of inadequate treatment response was significant, $\chi^2(5) = 16.90$, $p < .01$, $R^2_{\text{Nagelkerke}} = .29$. Similarly, a model testing comorbid frequent insomnia and headaches was also significant, $\chi^2(4) = 9.91$, $p < .05$, $R^2_{\text{Nagelkerke}} = .18$.

The efficacy of postgraduate psychologist trainees in reducing sleep and headache symptomatology, and overall psychiatric symptomatology were examined with non-parametric ranked comparisons, and paired t-tests. As single items on the OQ-45 are measured as ordinal data, a non-parametric test was used for assessing treatment effects of insomnia and headaches. For insomnia, a Wilcoxon signed ranked test indicated the frequency of insomnia at treatment termination was significantly lower relative to treatment onset, $Z = -3.12$, $p < .01$ (two-tailed), $r = .39$. Frequency of headaches at termination was significantly reduced compared to the initial sessions with a large effect, $Z = -4.34$, $p < .001$, $r = .53$. Table 4 presents initial and final session comparisons of all OQ-45 scales. The mean OQ-45 total score upon entering treatment at our clinic was 82.9 (SD = 18.84), and the mean at termination was 62.45 (SD = 25.65). A paired samples t-test revealed a large effect size for

treatments provided by our clinic interns $t(66) = 7.64, p < .001, d = 0.9$. Despite the large effect sizes, however, at treatment termination the prevalence of frequent insomnia was 32.8%, frequent headaches 14.9%, and frequent insomnia and headaches 9%.

Based on the prevalence of frequent insomnia and headaches, respectively, at treatment onset and termination, the number needed to treat (NNT) to revert frequent insomnia and headaches to “sometimes” or less was 6.7 (insomnia), 12 (headache), and 14.5 (comorbid insomnia and headache) patients.

Discussion

Prevalence data from our clinic revealed a more than two-fold prevalence of frequent insomnia (47.8%), compared to a community estimate of 20%. Frequent headaches occurred in only 23.2% of patients at treatment onset, compared to a community migraine prevalence of 25%. This discrepancy may be due to our study only examining frequent headaches, whereas the Deloitte study included episodic migraine. The correlation effect size, $r_s = .26$, found in our clinical sample between insomnia and headaches at treatment onset is a similar magnitude to our findings between poor sleep quality (measured by the Pittsburgh Sleep Quality Index) and headaches in a non-clinical Australian sample by Sullivan and Martin (2017), $r_s^{migraine} = .33, p < .001$; $r_s^{non-migraine\ headache} = .28, p < .001$. The effect size of this correlation found in the present study, however, is surprisingly small given the clinical population researched. However, this may be due to the different measurement approaches, with the PSQI measure in Sullivan and Martin (2017) encompassing a much broader variety of sleep pathology and consequences than the OQ item which is limited to insomnia only.

Significant correlations between insomnia and headaches and several OQ subscales at the initial session were found, and the presence of these issues frequently or greater at the initial session were associated with markedly increased odds of remaining significantly clinically distressed at treatment termination. This indicates that the presence of insomnia and

headaches may complicate other important psychosocial factors. One example is that headaches at the initial session correlated with the interpersonal relations subscale in the OQ-45. For example, it is conceivable that this may be due to common headache coping strategies, including napping, and trigger avoidance (such as insisting on a low light environment, or avoiding social gatherings), leading to conflicts within the familial or spousal unit. Whilst frequent insomnia and headaches, respectively, were independently associated with markedly greater odds of inadequate treatment response (remaining significantly distressed at termination), the presence of comorbid frequent insomnia and headaches at treatment onset (15.9% of the sample) was the strongest predictor of treatment failure found. There was a greater than 7-fold odds of treatment failure in those with comorbid insomnia and headaches than those who did not suffer from that combination of problems. The implication for clinical practice is that insomnia and headaches represent a treatable factor related to treatment failure in outpatient psychology patients. Our findings that headaches are predictive of poorer treatment outcomes are in line with a study by Hung, Liu, Yang, and Wang (2015) who found no psychiatric patients with an active migraine disorder achieved remission of a major depressive disorder within 2 years, compared to 25% of those without migraine, and 50% of those with an inactive migraine disorder.

In addressing RQ4, it was hypothesised that postgraduate trainee therapists would achieve similar effect sizes to those found in effectiveness studies of fully qualified therapists. Kraus et al. (2011) found the effect sizes of community outpatient therapists to range from $d = 0.27$ for sexual dysfunction, through to $d = 0.91$ for depression; effects on sleep were moderate, $d = 0.57$. These effect sizes are similar to those achieved by our student therapists, with an effect size of $d = 0.9$ in reducing the OQ-45 total. Effects on insomnia were greater in our student therapists, $d = 0.83$, than the effects on sleep in the sample of clinicians studied in Kraus et al. (2011). In a similar setting to our clinic, student therapists at

another Australian clinical psychology training clinic also achieved large effect sizes for face-to-face therapy, $d = 0.84$ (Brunnbauer, Simpson, & Balfour, 2016). Whilst student effect sizes were large, the Numbers Needed to Treat for insomnia, headaches, and comorbid insomnia and headaches remained relatively high. Given the students' effectiveness was comparable to community clinicians, the higher NNTs for insomnia and headaches are likely a product of the postgraduate clinical psychology program not including training in specific treatments for these issues, including CBT-i, and for headaches, Learning to Cope with Triggers (Martin, 2010). It is also possible that working on sleep and headaches were not therapy goals of some patients, even if these problems were occurring frequently. Whilst it is reassuring to demonstrate student therapists achieve large effect sizes, there are some confounding factors to be considered in comparing the outcomes of our student clinic with community outpatient clinics. These confounders mainly relate to the patient populations seen, whereby our student clinic routinely screens-out and refers-on intakes where there is a high risk of legal action (e.g., court-mandated therapy, insurance cases, and custody disputes), and individuals at chronic and high risk of suicide or harm to others.

Limitations to this study include the high proportion of patients at our clinic in 2018 whom did not consent to, or where we were unable to use their de-identified data for research purposes ($\approx 43\%$). As a result, most of the statistical analyses were underpowered. It is therefore unclear if many of the non-significant findings (particularly correlations) were true-negatives or were a result of a Type II error. Furthermore, the use of a broad outcome measure, as well as single items from that measure (for insomnia and headaches) does not allow for in-depth analysis, which may be useful and clinically important. Additionally, the OQ measure does not provide information on the patient's presenting problem, diagnosis, prescribed medications, or therapeutic approaches utilised (such as psychotherapeutic orientation or other medical treatments, for example, rTMS or ECT). As a retrospective

archival study, we were unable to compare therapy effect sizes to a control group, and therefore were only able to compare our effects to those published in previous studies. Future prospective research in this area may benefit from administering the OQ-45 to patients when they are added to the therapy wait-list in order to allow for comparisons to a control group.

In summary, we found that frequent insomnia and headaches are a highly prevalent problem in patients attending our clinical psychology training clinic in metropolitan Queensland. Despite first-year clinical psychology curricula generally not covering sleep or headache disorders, clinical psychology trainees achieved large effect sizes for these problems, likely due to generalisability of cognitive and behavioural strategies, and reduction of distress more generally. Frequent insomnia and headaches were associated with greater odds of psychological treatment failure, particularly when these issues were comorbid. As such, revision of clinical psychology programs across Australia to include content on sleep problems and headaches may be beneficial in improving treatment outcomes for other psychological presenting problems. Finally, the large treatment effect sizes for psychological distress broadly (OQ-45 total) indicates that GPs, psychiatrists, and the general public should be confident in referring-to and receiving treatment from clinical psychology trainees in the university clinic setting.

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Table 1

Spearman correlations between insomnia and headaches (initial session), and OQ-45 scales

	IF Initial	HF Initial	SD Final	IR Final	SR Final	OQ45 Initial	OQ45 Final
Insomnia Frequency – Initial Session	-						
Headache Frequency – Initial Session	.26*	-					
Symptom Distress – Final Session	.27*	.49**	-				
Interpersonal Relations – Final Session	.13	.42**	.67**	-			
Social Role – Final Session	.19	.45**	.79**	.60**	-		
OQ45 Total – Initial Session	.23*	.27*	.54**	.44**	.44**	-	
OQ45 Total – Final Session	.26*	.46**	.96**	.80**	.87**	.57**	-

** $p < .01$, * $p < .05$ – all comparison p values are one-tailed due to directional hypotheses

Abbreviations: IF – insomnia frequency, HF – headaches frequency, SD – symptom distress, IR – interpersonal relations, SR – social role, OQ – outcome questionnaire

Table 2

Characteristics of Patient and Therapist Sample

	N (% female)	Mean No. Sessions (SD, Range)	Mean Age Years (SD, Range)
Patients	69 (68.1%)	11.06 (SD: 8.016 Range: 1 – 36)	35.98 (SD: 14.72, Range: 18, 73)
	N (% female)	Mean Annual Caseload (Median, Range)	-
Therapists	19 (94.7%)	13.47 cases (Median = 13, Range = 7, 19)	-

Note: Mean annual caseload is based on the total caseload of therapists including adult, child, and couples therapy; and adult and child cognitive assessments.

Table 3

<i>Risk of Inadequate Therapy Response, Adjusted for Age, Sex, and Number of Sessions</i>				
Variable	B(SE)	Wald(df)	<i>p</i> (<i>Bootstrapped p</i>)	Odds Ratio (95% CI)
Model 1				
Sex (Female)	.96(.66)	2.10(1)	.15(.19)	2.62(.71, 9.61)
Age	.002(.02)	.01(1)	.92(.92)	1.00(.96, 1.05)
Number of Sessions	-.02(.04)	.19(1)	.66(.68)	.99(.92, 1.05)
Frequent Insomnia (Initial)	1.53(.58)	7.02(1)	.01(.01)	4.62(1.49, 14.35)
Frequent Headaches (Initial)	1.34(.70)	3.67(1)	.06(.04)	3.82(.97, 15.08)
Model 2				
Sex (Female)	.92(.62)	2.25(1)	.13(.14)	2.52(.75, 8.42)
Age	-.003(.02)	.02(1)	.88(.89)	1.00(.96, 1.04)
Number of Sessions	-.02(.03)	.23(1)	.63(.65)	.99(.92, 1.05)
Comorbid Frequent Insomnia and Headaches (Initial)	1.98(.86)	5.27(1)	.02(.01)	7.25(1.34, 39.34)

Note: Bootstrapped *p* values are two-tailed and based on 2000 bootstrap samples.

Table 4

Mean Scores on OQ-45 Scales at Initial and Final Session

OQ-45 Scale	Initial Session Mean (SD)	Final Session Mean (SD)	$t(df), p$	Effect Size d
Symptom Distress	49.36(12.16)	36.76(16.29)	7.21(66), $p < .001$	0.88
Interpersonal Relations	18.65(5.79)	14.33(6.68)	6.21(66), $p < .001$	0.68
Social Role	14.88(4.28)	11.36(5.26)	5.06(66), $p < .001$	0.71
OQ-45 Total	82.90(18.84)	62.45(25.65)	7.64(66), $p < .001$	0.90
