Cognitive Behavioural Therapy for Tinnitus-Related Insomnia: Evaluating a New Treatment Approach

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Abstract

Objective: Insomnia frequently occurs alongside distressing tinnitus, and greater tinnitus severity is associated with more sleep disturbance. Insomnia and tinnitus probably share common underlying processes and sleep studies show striking similarities between primary and tinnitus-related insomnia. This is the first study to evaluate outcomes following insomnia-specific Cognitive Behavioural Therapy (CBTi) for tinnitus-related insomnia in a ‘real world’ clinic.

Design: Treatment was six-sessions of group-based CBTi. Measures of insomnia, sleep diaries, tinnitus distress, psychological distress, anxiety and depression were completed pre-intervention, post-intervention and at six-weeks follow up.

Study sample: Participants were 24 adults with chronic, distressing tinnitus and associated sleep disturbance. Twenty-two completed treatment.

Results: CBTi was associated with significant improvements from pre-intervention to post-intervention maintained at follow up in insomnia, sleep-diary measures, tinnitus distress, psychological distress, anxiety and depression, largely maintained at follow-up. Reliable improvements were reported in insomnia (by 67% of patients), tinnitus distress (by 50% of patients) and psychological distress (by 38% of patients) post-intervention.

Conclusions: The results suggest that CBTi is associated with reduced insomnia and distress for patients reporting chronic and distressing tinnitus with related insomnia. Further research into CBTi for this population, using utilizing robust, randomized controlled designs, is warranted.
**Introduction**

Tinnitus is very prevalent, experienced by 10-15% of the population, with significant annoyance reported by 1-2% of the population (Baguley et al., 2013). Tinnitus is associated with emotional distress, sleep disturbance, and difficulties with auditory perception and concentration. Sleep disturbance is the most or second most common problem (Tyler & Baker, 1984; Sanchez & Stephens, 1997), reported by 50%-70% of people attending a tinnitus clinic (Jakes et al., 1985; Lindberg, et al., 1988). Insomnia in tinnitus has not been clearly defined in the literature (McKenna, 2000) but definitions of insomnia secondary to health conditions more generally may apply (Espie, et al 2008; Tang, et al., 2012).

A small amount of research has demonstrated a correlation between tinnitus and insomnia, although causal pathways have not been demonstrated. Tinnitus severity levels correlate with reported sleep disturbance (Schecklmann, et al., 2015; Folmer & Griest, 2000; Herbert et al., 2011) and quality of life. Polysomnography (PSG) studies show reduced sleep efficiency (SE) and total sleep time (TST) and increased sleep onset latency (SOL) and waking after sleep onset (WASO) in tinnitus patients, compared to controls (Burgos et al., 2005).

Research comparing patients with tinnitus-related insomnia to patients with primary insomnia have shown striking similarities in abnormalities in sleep architecture in both groups. Crönlein et al. (2007) found no significant differences between tinnitus-related insomnia and primary insomnia patients on objective (EEG/Electroencephalogram, EOG/Electroculogram, EMG/Electromyogram, respiration), or subjective (SE) measures, except that tinnitus patients had longer subjective SOL. Daytime levels of attention, tiredness and depression rating scores were also equivalent. Similar observations were reported by Burgos et al. (2005), and both tinnitus-related and primary insomnia groups, reported more subjectively impaired
sleep, decreased SE, TST and increased frequency of night time awakenings than healthy controls. Additionally, tinnitus patients had increased SOL compared to the other groups and a decreased percentage of stage 2 sleep and elevated Rapid Eye Movement (REM) sleep density and percentage compared to controls.

When tinnitus-related insomnia patients are compared to healthy controls, differences in sleep architecture have been reported. Attanasio, et al. (2013) found that tinnitus patients had less deep (stage 3, 4) and REM sleep than healthy controls, and there was a positive correlation between lighter sleep and self-reported tinnitus distress. Herbert, et al. (2011) found that tinnitus patients had reduced subjective sleep quality, and reduced TST and although this was not supported by PSG, they found lower EEG delta power during Non-Rapid Eye Movement (NREM) sleep in tinnitus patients. A few studies, using self-report questionnaires, found that tinnitus patients reported poorer sleep quality, increased insomnia-specific concerns and more anxiety than healthy controls (Crönlein et al., 2016). Overall findings suggest that sleep in tinnitus-related insomnia is similar to primary insomnia and different from healthy controls.

Researchers have suggested that tinnitus and insomnia may share similar underlying processes, including autonomic hyperarousal, reduced serotonin and depression (Wallhäusser-Franke et al., 2013; Attanasio et al. 2013; Burgos, et al., 2005). Both conditions are associated with high stress levels and so may be triggered by similar life events. Alternatively, tinnitus itself may cause insomnia, and clinical observation has found this attribution is commonly made by patients. Once triggered, it is possible that tinnitus-related insomnia is maintained in the same way as primary insomnia (by cognitive, behavioural, attentional and emotional changes). In this case, it may be reasonable to assume that
treatments for primary insomnia can apply to tinnitus-related insomnia (Crönlein, et al., 2007).

Evidence for psychologically-based treatments for tinnitus-related insomnia is scarce. Systematic reviews of interventions for tinnitus have not included sleep disturbance as an outcome and no trials have specifically investigated tinnitus patients reporting clinical levels of insomnia. Interventional research including sleep disturbance as an outcome has used poor quality measures or small groups (Lindberg et al., 1989; Kroner-Herwig, et al. 1995; Andersson, et al., 2005). Studies of non-sleep specific CBT has shown improvement on sleep diaries, a visual analogue scale and sleep subscale of the Tinnitus Questionnaire (Weise, et al., 2008; Kaldo, et al., 2007; Seydel, et al., 2010). Two internet-based trials of CBT have found that insomnia improves using a standardized measure of insomnia (Weise et al., 2016; Jasper et al., 2014)

Cognitive Behavioural Therapy for Insomnia (CBTi) has proven efficacy as treatment for primary insomnia with with medium to large effect sizes reported by high-quality systematic reviews (Okajima, et al 2011; Morin, et al., 2006). Encouragingly, evidence has shown that CBTi is equally effective for insomnia secondary to another health condition (Morin, et al., 2006), including cancer (Espie, et al., 2008; Savard, et al., 2005) and chronic pain (Tang, 2009; Jungquist, et al 2010; Jungquist, et al., 2012; Tang et al 2012).

Considering the similarities between tinnitus and chronic pain (Møller, 2007), the findings in the pain literature are of particular interest. For example, CBTi leads to improved sleep and pain-interference in the long-term, regardless of pain severity (Jungquist, et al., 2010; Jungquist, et al., 2012). Tang (2009) argues that, since sleep deprivation reduces tolerance for
persistent physical symptoms, and is maintained by independent cognitive-behavioural factors (Tang et al., 2012), secondary insomnia requires direct intervention in order to be resolved. If pain-related insomnia responds to CBTi, tinnitus-related insomnia may do too, but no studies have yet tested this. Our clinic routinely offers cognitive-behavioural therapies for tinnitus, and when sleep is a primary issue, we offer CBTi for tinnitus.

**Aims and Hypotheses**

This (uncontrolled) study aimed to evaluate the outcomes associated with group-based CBTi for patients reporting tinnitus-related insomnia, using reliable change on standardized measures as criteria for improvement.

We hypothesised that:

1. CBTi would be associated with a reliable and clinically significant reduction in sleep disturbance, as measured a standardised insomnia questionnaire, and statistically significant improvements in sleep, as measured by sleep diaries.
2. CBTi would be associated with a reliable and clinically significant reduction in tinnitus-related distress and general psychological distress.
3. CBTi would be associated with statistically significant reductions in levels of anxiety and depression.

**Methods**

**Participants**

As this was an evaluation of routine clinical work, eligibility criteria were as follows: Participants were 24 adults reporting chronic distressing tinnitus (for six months or longer), and sleep disturbance, who had completed medical/audiological examinations, and had been
referred to psychology for help with tinnitus. Many reported clinical levels of anxiety and/or depression, but this did not affect treatment offered. Exclusion criteria included individuals reporting high levels of risk (suicidality, substance misuse or self-harm), or a preference against group treatment; individual therapy was offered to people excluded on these grounds.

From March 2016 to April 2017, 204 tinnitus patients were referred to the psychology service. Of these, 17 were referred specifically with tinnitus-related insomnia and seven others reported insomnia and a preference for sleep-treatment at assessment. All other patients were deemed more appropriate for tinnitus-focused (rather than sleep-focused) therapy.

**Procedures**

Patients referred were assessed by a clinical psychologist within four months (according to a routine waiting list). Assessment included clinical interview and standardized outcome measures. If significant sleep disturbance was reported, patients were given information about the CBTi group and could opt for this or alternative psychological therapy. Waiting time from assessment to CBTi varied from one to eight weeks. Three clinical psychologists offered CBTi, working in pairs (EM / LM or EM / FV). The treatment manual had been developed as part of routine service development, and adherence to the manual/treatment integrity was managed via regular team meetings. Treatment completion was regarded as attending at least 50% (3 treatment sessions).

**Intervention**

CBTi for tinnitus was based on typical CBTi, with six sessions involving sleep diary monitoring, time-in-bed restriction, stimulus control, relaxation training, cognitive
restructuring and behavioural change. Additional tinnitus-focused elements we also included: Psychoeducation about tinnitus, understanding shared processes in tinnitus and insomnia, and provision of a night-time sound machine.

**Measures**

Routine, standardized outcome questionnaires were completed three times (pre-intervention; post-intervention; six-week follow up). A sleep diary was kept every night from 2-weeks before session 1 until the final follow up. Demographic information was routinely collected.

**Primary Clinical Outcome (Insomnia Severity Index)**

**Insomnia Severity Index (ISI), patient version** (Bastien, et al., 2001): A 7-item retrospective self-report questionnaire on a 5-point Likert scale assessing the nature, severity, and impact of insomnia over two weeks. Total scores are: No insomnia (0–7); sub-clinical insomnia (8–14); moderate insomnia (15–21); severe insomnia (22–28). It has excellent internal consistency in patient samples (Cronbach α 0.91), is sensitive to treatment response, and can show the clinical significance of change (whilst sleep diaries alone can only show statistical significance) (Morin et al., 2011). A 6-point reduction has been recommended as showing clinically meaningful change in primary insomnia (Yang et al., 2009).

**Secondary Clinical Outcomes**

**Sleep Measures (Diaries):** Sleep diaries are an effective measure of statistically significant changes in sleep. Patients recorded the following every day: Sleep Onset Latency (SOL), Wake-time After Sleep Onset (WASO), Total Sleep Time (TST), Time spent In Bed (TIB). From this a key measure of insomnia, Sleep Efficiency (SE) is calculated (SE = TST / TIB x 100).
**Tinnitus-related Distress:** The Tinnitus Questionnaire (TQ) is a self-report questionnaire with 41 items scored 0 – 2 (Hallam, 1996). A reliable change index (RCI) of 11.08 was used. This had been established previously in our population using the Jacobson & Truax formula (1991) (see McKenna, Marks & Vogt, 2017). There is high test-retest reliability (r =.94) and internal consistency (α =.93) (Hiller et al., 1994).

**General Psychological Distress:** The Clinical Outcome in Routine Evaluation - Outcome Measure (CORE-OM) (Evans, et al., 2000) assesses general psychological state through 34-items rated on a 0 to 4 scale. It can be scored as two scales: “risk” and “psychological distress”. The non-risk scale (CORE-NR) is the mean of the 28 non-risk items multiplied by 10 (McKenna et al., 2017). An average score of 10 or more is ‘clinically significant’ and the reliable change criterion is ≥5. When a subject’s score decreases by ≥5 and moves from the clinical to the non-clinical range (i.e. from > 10 to < 10) the individual is regarded as being reliably and significantly improved. It is an appropriate measure of distress in tinnitus patients (Handscomb et al., 2016).

**Depression:** The Patient Health Questionnaire-9 (PHQ-9) has nine items on a 0 to 3 scale, measuring depressive symptoms over the preceding two weeks (Kroenke, et al., 2001). Total scores range from 0 to 27. Scores of 10 and above indicate the presence of clinical depression.

**Anxiety:** The GAD7 assessed anxiety via seven items rated on a similar 0 to 3 scale as the PHQ9 (Spitzer, et al., 2006). Total scores range from 0 to 21 and scores of 8 and above indicate the presence of clinical anxiety.

**Ethics**

The Hospital Trust gave permission for publication of this report as an evaluation of routine clinical work.
Statistical analyses

Summaries are presented as means [SE] and n (%), as appropriate. Repeated measures ANOVA with Bonferroni corrections were used to assess the primary outcome of Insomnia Severity (ISI) and secondary outcomes of Sleep Efficiency (SE), Sleep-onset latency (SOL), Wakefulness after sleep-onset (WASO), Total Sleep Time (TST), tinnitus distress (TQ), psychological distress (CORE-OM), Anxiety (GAD-7) and Depression (PHQ-9). Comparison of pre-post and pre-follow-up was made separately. Effect sizes for repeated measures designs are Cohen’s d, calculated with Morris (2008).

Results

Of the 24 patients included, 22 (92%) completed treatment (two dropped out, one due to illness, one due to other demands). Fifteen attended follow-up. Of completed questionnaires, missing data was minimal on outcome variables (<4%). Data was missing for seven participants at the final session and for nine at follow-up (due to non-attendance of treatment completers). Missing data was consistent with the assumption of Missing at Random (MAR) and hence handled using multiple imputation (MI) using primary outcome and process variables (Ware, et al. 2012).

Patient characteristics

Participants’ mean age was 55.71 [12.14] years (range 35 to 79). Gender was equally distributed (50% male). All patients had chronic tinnitus (present for at least three months) by the commencement of therapy, with an average of 23.17 months (range 4 to 42 months). The average group size was 8 people (range 7 to 9). Baseline mean TQ score was (46.25 [3.05]), in the third quartile of scores observed in a tinnitus clinic (moderate to severe distress). Table
1 summarizes treatment effect on primary outcomes and Table 2 summarizes treatment effect on secondary outcomes.

<INSERT TABLE 1 NEAR HERE>

Table 1. Effect of treatment on primary outcomes: Insomnia and sleep diaries

**Primary Outcomes**

Repeated-measures ANOVAs showed improvements across a range of insomnia measures:

**Insomnia Severity Index (ISI):** Levels of insomnia changed across the intervention. It decreased significantly from pre- to post-intervention, by 10.1 points (p<.01) and maintained a reduction at follow-up (p<0.05). Sixty-seven percent (66.7%) experienced reliable improvement (i.e. a reduction ≥ 6) from pre- to post-intervention, and 54.2% from pre-intervention to follow-up.

**Sleep efficiency (SE)** increased significantly during the intervention. Sleep efficiency increased by 12.7% from pre- to post-intervention (p<0.01) and maintained a similar improvement at follow-up (p<0.05).

**Sleep-onset latency (SOL)** changed significantly during the intervention. Participants fall asleep 11.7 minutes sooner post-intervention, but the difference was not significant. By follow-up sleep onset reduced by a further 8.5 minutes compared to post-intervention, which was significant (p<0.01).

**Wakefulness after sleep-onset (WASO)** changed significantly across the intervention. It decreased post-intervention by 20.9 minutes (p<0.05). However, follow-up wakefulness was not significantly different to either pre-intervention or post-intervention (p>.05).

**Time in bed (TIB)** changed significantly as part of the intervention. It decreased by 60.5 minutes from pre- to post-intervention (p<0.01) but increased again by 35.1 minutes at
follow-up (p<0.01) compared to post-intervention.

Total sleep time (TST) did not change significantly during the intervention.

Secondary Outcomes

Tinnitus-related distress (TQ): Tinnitus distress decreased significantly from pre- to post-intervention, maintained at follow up, (see table 2) demonstrating sustained significant improvement. Fifty percent experienced reliable improvement (i.e. a reduction >11.08) from pre- to post-intervention, and 41.7% from pre-intervention to follow-up.

Psychological distress (CORE-NR): Psychological distress decreased significantly from pre to post-intervention and to follow-up (see table 2). Reliable improvement post-intervention was observed in 37.5% of participants, and by 41.7% at follow-up. Of those who were in a clinically significant state of psychological distress (i.e. CORE>-NR >10) before the intervention, 41.7% were in a non-clinical range post-intervention and 41.1% were in a non-clinical range at follow-up. Both reliable change and moving into remission (i.e. from above to below the clinical cut-off point of 10) was reported by 29.2% of the sample post-intervention and 25% of the sample by follow-up.

Anxiety and Depression (PHQ9, GAD7): The same pattern of reduced symptomatology was found on specific measures of anxiety and depression (see table 2). Both depression (PHQ9) and anxiety (GAD7) decreased significantly from pre- to post-intervention, but this decrease was only sustained for depression.

<INSERT TABLE 2 NEAR HERE>

Table 2. Effect of treatment on secondary outcomes: Tinnitus and Psychological Distress
Discussion

This is the first ever study to report on outcomes associated with insomnia-specific CBT (CBTi) for patients with tinnitus-related insomnia. As hypothesized, CBTi was associated with improvement across a range of well-established sleep outcomes, with 66.7% reporting reliable improvement on the ISI post-treatment, sustained by 54.2% at follow-up. There were also statistically significant improvements seen in SE and SOL, TIB and WASO alongside significant reductions in tinnitus-related and psychological distress, anxiety and depression.

Findings are clinically important in light of the evidence that sleep disturbance is prevalent in tinnitus, with delayed Sleep Onset Latencies (SOL), increased Wake Time After Sleep Onset (WASO), reduced Sleep Efficiency (SE) and Total Sleep Time (TST) (Burgos et al., 2005; Cronlein et al., 2016). The association of CBTi with changes on all of these aspects of sleep (excepting TST and WASO at follow-up) is encouraging, as are the findings of moderate-to-large effect sizes in the primary outcomes of interest (ISI, SOL and SE). This is in line with trials of CBTi for insomnia that is primary, or secondary to other health conditions, including chronic pain. Meta-analyses tend to report effect sizes as greater for SOL than TST (e.g. Harvey & Tang, 2003), and single RCTs of CBTi for illness-related insomnia report significant improvements in SE, SOL and WASO, but non-significant increases in TST (e.g. in chronic pain – Jungquist et al., 2010; cancer - Espie, et al., 2008). This may be because longer follow-up periods are required to see change in TST, as CBTi focuses on sleep consolidation before increasing sleep time.

Patients in this evaluation also reported significantly lower tinnitus-related and psychological distress following treatment. Reliable improvements in tinnitus distress were reported by
50% of patients and in psychological distress by 37.5% of patients post-intervention, with such changes largely sustained by follow-up. Again, this indicates potential clinical importance, with the percentage of improvement in line with other tinnitus-specific psychological treatments, including those offered by our clinic, such as Mindfulness Based Cognitive Therapy for tinnitus (McKenna, Marks & Vogt, 2017; McKenna et al., 2017). Improvement was also seen in anxiety and depression, although only maintained in depression. Improvements in multiple domains are noteworthy for two reasons: Insomnia is associated with more severe tinnitus (e.g. Schecklmann, et al., 2015; Folmer & 2000; Herbert, et al., 2011); and tinnitus can be problematic even without sleep disturbance (Baguley, et al., 2013). Any treatment in a clinical setting should be able to improve tinnitus and distress as well as sleep, and these initial findings for CBTi warrant further investigation in this area.

CBTi has been thoroughly tested and described across the literature, and found to be acceptable and replicable (Harvey & Tang, 2003). This appeared to be reflected in our high (92%) completion rates. Attendance at a follow-up appointment was lower, possibly since this additional session was a ‘booster’ rather than further treatment, potentially reducing motivation to attend. As our treatment follows established CBTi protocols, it should be easily replicable, with appropriate training and supervision.

Strengths of this study are the naturalistic design with minimal exclusion criteria and all patients reporting chronic tinnitus and insomnia, severe enough to seek treatment at a tertiary care level. There are also important limitations. It is a non-controlled evaluation of routine clinical practice, with a small sample size, missing data and a relatively short follow-up period of 6 weeks. There may also be a source of bias, with self-reported data being collected
as part of routine care, by the treating clinicians and not by independent researchers. To minimize bias, most data-entry was completed by a team member not involved in treatment delivery. It is noteworthy that, although tinnitus can improve over time (e.g. during a waiting period; Hesser et al., 2011), improvements are unlikely to be of the sizes seen here over just 8 weeks (McKenna, et al., 2017). Similarly, chronic insomnia rarely improves spontaneously (Tang, 2009). Without a control group, it is difficult to ascertain the specific effects of CBTi, but the moderate to large effect sizes across measures, and sizeable percentages of patients showing reliable improvement are encouraging, and in line with other tinnitus treatments previously tested within our population (McKenna, Marks & Vogt, 2017; McKenna et al., 2017). Even so, we recommend that these results be interpreted with caution.

**Implications**

This study offers a new addition to the literature on tinnitus and insomnia. CBTi was associated with improved sleep, tinnitus and distress, in line with theory and evidence for tinnitus-related insomnia. This finding adds to evidence that insomnia secondary to tinnitus may be phenomenologically and psychologically similar to primary insomnia, and so responsive to insomnia-focused cognitive and behavioural treatment.

The findings could have meaningful clinical implications, and it is clearly necessary to test this treatment approach in a rigorous way with larger samples and randomized controlled design. Only when CBTi is compared to control groups, including currently available treatment, will it be possible to say whether this treatment leads directly to the improvements shown here. Further research is warranted considering the pernicious and debilitating effects of insomnia-related tinnitus. As this study tentatively suggests, it may be possible for tinnitus patients with insomnia to develop better sleep through the process of CBTi.
Disclosure Statement

The authors report no conflicts of interest. No funding was provided for this study.

Geolocation information

This study was based in London, UK.

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References


35. Morin CM; Belleville G; Bélanger L; Ivers H. The insomnia severity index: psychometric indicators to detect insomnia cases and evaluate treatment response. SLEEP 2011;34(5):601-608.


Table 1. Effect of treatment on primary outcomes: Insomnia and Sleep Diaries

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-Intervention</th>
<th>6-week follow-up</th>
<th>ANOVA, F, p-value</th>
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</thead>
<tbody>
<tr>
<td>ISI, mean [SE]</td>
<td>20.2 [0.9]</td>
<td>12.1 [1.1]</td>
<td>12.3 [1.2]</td>
<td>(2, 46) 31.780,  p&lt;.01</td>
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<tr>
<td>Effect Size – Cohen’s d</td>
<td>-</td>
<td>d=1.36</td>
<td>d=1.19</td>
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<tr>
<td>Reliable Improvement (reduction ≥ 6) n (%)</td>
<td>66.7% (16)</td>
<td></td>
<td>54.2% (13)</td>
<td></td>
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<tr>
<td>Effect Size – Cohen’s d</td>
<td>-</td>
<td>d=0.80</td>
<td>d=0.62</td>
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<tr>
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<td>d=0.96</td>
<td>d=0.31</td>
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<td>-</td>
<td>d=0.03</td>
<td>d=0.21</td>
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<tr>
<td>SOL, mean [SE]</td>
<td>45.4 [6.5]</td>
<td>33.7 [6.8]</td>
<td>25.2 [4.2]</td>
<td>(2, 46) 6.026,  p&lt;.01</td>
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<tr>
<td>Effect Size – Cohen’s d</td>
<td>-</td>
<td>d=0.42</td>
<td>d=0.59</td>
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<tr>
<td>WASO, mean [SE]</td>
<td>46.5 [7.0]</td>
<td>25.6 [5.5]</td>
<td>32.3 [6.1]</td>
<td>(2, 46) 4.865,  p&lt;.05</td>
</tr>
<tr>
<td>Effect Size – Cohen’s d</td>
<td>-</td>
<td>d=0.49</td>
<td>d=0.34</td>
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</table>

n.s. = not significant at p >0.05; a b Time points with similar superscripts do not differ (p > 0.05).
Table 2. Effect of Treatment on Secondary Outcomes: Tinnitus and Psychological Distress

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-intervention</th>
<th>6-week follow-up</th>
<th>ANOVA, F, p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tinnitus distress, mean [SE]</strong></td>
<td>46.2 [3.0] a</td>
<td>32.1 [2.8] c</td>
<td>36.2 [2.8] b</td>
<td>(2, 46) 13.749, p&lt;.01</td>
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<tr>
<td>Effect Size to Pre-intervention – Cohen’s d</td>
<td>-</td>
<td>0.91</td>
<td>0.62</td>
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<tr>
<td>Reliable Improvement (reduction ≥ 11.47%) (%)</td>
<td>-</td>
<td>50.0% (12)</td>
<td>41.7% (10)</td>
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<tr>
<td><strong>Psychological distress, mean [SE]</strong></td>
<td>14.7 [1.4] a</td>
<td>10.8 [1.3] b</td>
<td>10.6 [1.3] b</td>
<td>(2, 46) 7.86, p&lt;.01</td>
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<tr>
<td>Effect Size to Pre-intervention – Cohen’s d</td>
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<td>0.72</td>
<td>0.62</td>
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<tr>
<td>Reliable Improvement (reduction ≥ 5%) (%)</td>
<td>-</td>
<td>37.5% (9)</td>
<td>41.7% (10)</td>
<td></td>
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<tr>
<td>Remission (reduction from &gt;10 to ≤10%) (%)</td>
<td>-</td>
<td>41.7% (10)</td>
<td>37.5% (9)</td>
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<tr>
<td>Reliable recovery (reliable improvement and remission) (%)</td>
<td>-</td>
<td>29.2% (7)</td>
<td>25% (6)</td>
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<td><strong>Symptoms depression, mean [SE]</strong></td>
<td>12.3 [1.1] a</td>
<td>7.6 [1.2] b</td>
<td>8.6 [1.1] b</td>
<td>(2, 46) 9.751, p&lt;.01</td>
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<tr>
<td>Effect Size to Pre-intervention – Cohen’s d</td>
<td>-</td>
<td>0.93</td>
<td>0.58</td>
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<tr>
<td><strong>Symptoms Anxiety, mean [SE]</strong></td>
<td>9.5 [0.97] a</td>
<td>6.7 [1.2] b</td>
<td>7.3 [1.1] a</td>
<td>(2, 46) 5.353, p&lt;.01</td>
</tr>
<tr>
<td>Effect Size to Pre-intervention – Cohen’s d</td>
<td>-</td>
<td>0.69</td>
<td>0.43</td>
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</table>

n.s. = not significant at p >0.05; a b Time points with similar superscripts do not differ (p > 0.05).