Behavioral management of the triggers of recurrent headache: A randomized controlled trial

Behavioral Management of the Triggers of Recurrent Headache: A Randomized Controlled Trial

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Abstract

This study was designed to evaluate the traditional advice to headache sufferers to avoid all triggers ('Avoidance'), and a novel approach to trigger management (*Learning to Cope with Triggers* – 'LCT') that included graduated exposure to selected triggers to promote desensitization. Individuals (84F, 43M) with migraine and/or tension-type headache were assigned randomly to one of four groups: Waiting-list (Waitlist); Avoidance; Avoidance combined with cognitive behavior therapy (Avoid + CBT); and LCT. Changes in headaches and medication consumption (in parentheses) from pre- to post-treatment were (a minus sign indicates improvement): Waitlist, +11.0% (+15.4%); Avoidance, -13.2% (-9.0%); Avoid + CBT, -30.0% (-19.4%); and LCT, -35.9% (-27.9%). Avoidance did not differ significantly from Waitlist on headaches or medication use, but LCT differed significantly from Waitlist on both measures. Avoid + CBT significantly differed from Waitlist on headaches but not medication consumption. In summary, the study failed to find support for the standard approach to trigger management of advising avoidance, but LCT emerged as a promising strategy. LCT resulted in greater improvement than the other three conditions on all measures of headaches and medication consumption, and was the only treatment condition that significantly differed from the waiting-list control condition in terms of treatment responder rate (50% or greater reduction in headaches) and medication consumption.

Keywords: migraine, tension-type headache, triggers, desensitization, coping

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In a study of the triggers of migraine attacks, Kelman (2007) found 76% of migraineurs reported triggers when asked, and this figure rose to 95% when individuals responded to a specific list of triggers. Inspection of seven recent studies (Andress-Rothrock, King, & Rothrock, 2010; Deniz, Aygul, Kocak, Orhan, & Kaya, 2004; Ierusalimschy & Moreira Filho, 2002; Karli, Zarifoglu, Calisir, & Akgoz, 2005; Kelman, 2007; Leone, Vila, & McGowan, 2010; Spierings, Ranke, & Honkoop, 2001) suggests that the most common headache triggers are: (i) stress and negative emotions; (ii) sensory triggers (flicker, glare, eyestrain, noise, odours); (iii) hunger; (iv) lack of sleep or excess of sleep; (v) food and drink (particularly chocolate, cheese), and alcohol; (vi) menstruation; and (vii) weather (cold, heat, high humidity). Many other factors have been listed including exercise, fatigue, sexual activity, smoke, head and neck movements. A number of studies have investigated whether migraine and tension-type headache (TTH) have the same or different triggers, and most have failed to find differences (Chabriat, Danchot, Michel, Joire, & Henry, 1999; Philips & Hunter, 1981; Scharff, Turk, & Marcus, 1995), but some more recent studies suggest that differing triggers may be associated with each type of headache. Light, odours, hunger, weather and smoke have been reported as more common triggers in migraine than TTH, with head and neck movements more common in TTH (Karli et al., 2005; Spierings et al., 2001).

Interest in the triggers of headaches has been motivated by the idea that if triggers can be avoided, then headache frequency should be reduced. Advice to identify and avoid triggers as a means of preventing headaches has been the standard for decades. Researchers regularly make this point, for example, claiming that “migraine prevention is best achieved by avoidance of known migraine triggers” (Skaer, 1996, p. 229), and “comprehensive migraine treatment programs emphasize awareness and avoidance of trigger factors as part of the therapeutic regimen” (Friedman & De Ver Dye, 2009, p. 941). One of the ‘seven elements of

good headache management' listed by WHO is "identification of predisposing and/or trigger factors and their avoidance through appropriate lifestyle change" (WHO, 2006, p. 77). This advice appears on numerous internet sites. For example, the American Headache Society web site includes a section entitled 'Trigger Avoidance Information'. Headache apps are now available for smart phones that encourage trigger avoidance (e.g., *iManage Migraine*, Merck & Co). Surveys demonstrate that trigger avoidance is a widely used strategy by migraineurs (e.g., Peters et al., 2005).

There is surprisingly little empirical evidence supporting this approach, however. The best and most cited supportive study encouraged migraineurs to avoid all precipitating factors and reported a reduction of 50% in headache frequency in 19 out of 23 patients (Blau & Thavapalan, 1988). However, methodological shortcomings included no control conditions, and comparing a retrospective estimate of attack frequency in the three months before consultation with 'noting attacks' during the two months after consultation. Also, patients were advised on "how to abort attacks by quickly taking an antinauseant and analgesic tablets", and so advice about how to more effectively use medication was confounded with advice to avoid precipitants.

Recently, three reviews have presented arguments against advising avoidance of all headache triggers (Martin & MacLeod, 2009; Martin, 2010a, 2010b). As pointed out in these reviews, one problem with advising trigger avoidance is that the mechanisms that link triggers to headaches are not entirely clear. While sensitivity to triggers may have a genetic component (Bussone, 2004), if restricted prior exposure to triggers also is a contributing factor, then encouraging avoidance may increase trigger potency. Another problem is that from a practical perspective, it is not possible to completely avoid all potential headache triggers as they are so diverse; and attempting to do so could result in a restricted lifestyle (Kelman, 2007). Marcus (2003) pointed out that the effort to avoid every potential headache trigger may itself be stressful and so could exacerbate stress-related headaches. A third

potential problem is that advising trigger avoidance may lead to reduced internal locus of control for headaches, with attendant adverse effects on self-efficacy, particularly concerning one's perceived capacity to cope effectively with triggers (Marlowe, 1998).

Further arguments against advice to avoid triggers arise from consideration of cognate literatures. In the chronic pain literature, fear-avoidance models have been developed, which contend that individuals who confront their pain are considered more likely to adaptively resume physical and social activities, whereas those who respond to pain with avoidance are considered more likely to enter a self-perpetuating vicious cycle that maintains and exacerbates pain perception, leading to chronic pain and related disability (e.g., Asmundson, Norton, & Vlaeyen, 2004; Vlaeyen & Linton, 2000; Crombez, Eccleston, Van Damme, Vlaeyen, & Karoly, 2012). These models have led to research investigating interventions that target pain-related fear including exposure, and some successes have been reported (Leeuw, Goossens, Linton, Crombez, Boersma, & Vlaeyen, 2007). Parenthetically, Nash, Williams, Nicholson and Trask (2006) presented evidence that pain-related anxiety may have an important role in contributing to disability in headache sufferers. In the stress literature, it has been pointed out that coping with stress generally takes one of two routes – avoidance or approach, and the research evidence demonstrates that the avoidance coping pathway is not adaptive, with a few important exceptions (Snyder & Pulvers, 2001). Hayes et al. (2004) similarly conclude that higher levels of 'experiential avoidance', a type of avoidant coping, are associated with higher levels of general psychopathology and a lower quality of life.

The anxiety literature has demonstrated that short exposure to anxiety-provoking stimuli can increase subsequent anxiety responses to these stimuli, but prolonged exposure results in decreased subsequent anxiety responses (Eysenck, 1979). It is short exposure, resulting from attempts to avoid, or escape from, anxiety-eliciting situations, that underlies the maintenance of fears and phobias. Therapeutic approaches that involve prolonged exposure to anxiety triggers have been used with great success to treat a wide range of anxiety disorders (Barlow,

2004). Moses and Barlow (2006) have proposed a unified treatment approach for emotional disorders which includes preventing behavioral avoidance and cognitive avoidance in order to increase emotional exposure and attenuate the potency of emotional triggers. Driven by an analogy between the triggers of anxiety and the triggers of headaches, a series of studies have demonstrated that short exposure to some headache triggers (visual disturbance, noise, stress) leads to sensitization to the triggers and prolonged exposure leads to desensitization (Martin, 2001; Martin, Reece, & Fordyce, 2006; Martin, Lae, & Reece, 2007; Martin, 2000).

The reviews proposed that counselling avoidance of all triggers should be replaced with a philosophy of *Learning to Cope with Triggers* (LCT) (Martin & MacLeod, 2009; Martin, 2010a, 2010b), whereby triggers that are potentially harmful to health and well-being should be avoided, but for other triggers planned exposure should be used to promote desensitization and increased tolerance for triggers. The word 'cope' is used because of the insights that can be derived from the stress literature which demonstrates that no single coping strategy can be selected as the best way of coping with stress for all situations and across time, but shows that approach strategies generally are more adaptive than avoidance strategies (Suls & Fletcher, 1985). Martin and colleagues have argued that similarly, no one strategy can be singled out as the best way of managing all headache triggers. Sometimes avoidance will be the strategy of choice but approach/engagement/exposure strategies often will be more effective.

The study reported here was a randomized controlled trial designed to evaluate the standard approach to trigger management of avoidance on the one hand, and the novel approach of LCT on the other. The two interventions were compared with a waiting-list control condition. Advice to avoid headache triggers would usually be given in a relatively brief time frame and so it was delivered across three 30-minute sessions in the present study. LCT was delivered across eight 60-minute sessions. A second control condition was included in which the trigger avoidance approach was supplemented by cognitive behavior therapy (CBT), to match the number and duration of sessions in the LCT condition.

Methods

Participants

Inclusion criteria: (i) diagnosed as either 'typical aura with migraine headache', 'migraine without aura', 'chronic migraine', 'frequent episodic tension–type headache', or 'chronic tension-type headache' (Headache Classification Subcommittee of IHS, 2004); (ii) minimum of 6 headache days per month; (iii) minimum headache chronicity of 12 months, and pattern of headache symptoms stable over last 6 months; and (iv) aged 18 years and over.

Exclusion criteria: (i) pregnant, planning pregnancy during trial period or lactating; (ii) substantial medical or psychiatric co-morbidities that are deemed likely to interfere with ability to fully participate (determined by clinical judgement); and (iii) currently taking headache prophylactic medication (3-month wash-out).

A number of different recruitment strategies were used: general practice referrals; the media; posters; websites or newsletters of various organizations; and YouTube and Facebook.

Participant flow through the study is summarized in the CONSORT Flow Diagram in Figure 1. Demographic and headache characteristics of the sample are shown in Table 1. One type of headache was identified for 66 participants and two types of headaches for 61 participants. The diagnoses in Table 1 are the primary diagnoses.

The plan for the study based on power analysis was to recruit a sample of 180 participants, but the funding provided was less than requested and it was only possible to randomize 127 participants on this basis. Participant recruitment commenced in February 2010. The final participants were recruited in mid-2011 with follow-up completed in September, 2012.

A χ^2 analysis found no significant association between treatment group and either headache diagnosis (migraine versus TTH) or gender, and ANOVA found no significant difference in age or chronicity between the groups.

Independent groups *t*-test comparison of the 67 participants who completed the 12-month follow-up and the 60 participants who did not (either because they were in Waitlist or dropped-out of treatments groups at various stages of the protocol), revealed no significant differences on any of the baseline variables. Nor was there any evidence from χ^2 analysis that rate of drop-out from treatment was related to gender, headache diagnosis or treatment group.

Design

The study employed a mixed design, consisting of one between-subjects factor (Group), and one within-subjects factor (Time). A stratified randomization procedure based on the CONSORT guidelines was used to allocate participants to the four groups. Stratification within groups was on the basis of diagnosis (i.e., migraine versus TTH).

The study was approved by the Monash University Human Research Ethics Committee (HREC Number: CF09/0964-2009000447). The study was registered with the Australian New Zealand Clinical Trials Registry (ACTRN12610000393055).

Measures

Headaches and medication consumption. Participants completed diaries by recording hourly ratings of headache intensity throughout the waking day using a scale from 0 (*no headache*) to 5 (*an intense incapacitating headache*). This 'time-sampling' procedure has well-established reliability (Collins & Thompson, 1979) and validity (Blanchard, Andrasik, Neff, Jurish, & O'Keefe, 1981), and is generally regarded as the 'gold standard' in behavioral headache research (Andrasik, Lipchik, McCrory, & Whitrock, 2005). Medication consumption was also recorded in daily diaries (type, dosage and time taken). As these daily diaries result in a large data set, we devised a system using KeyPoint version 5.5 (Cambridge Software Publishing) to electronically scan diaries and export the data to an SPSS file.

Avoidance of triggers. The Trigger Specific Avoidance Scale of the Headache Triggers Avoidance Questionnaire (HTAQ) was used to assess trigger avoidance. This Scale has 24 items and measures how often respondents try to avoid the factors that trigger their

headaches. It has good internal consistency (Cronbach alpha of .81), good test-retest reliability over 3 to 4 weeks (r = .90), and construct validity shown via significant correlations with related scales such as the Anxiety Sensitivity Index and Pain Anxiety Symptoms Scale (Wood & Martin, in preparation).

Self-efficacy and locus of control. The Headache Management Self-Efficacy Scale (**HMSES**) consists of 25 items that assess an individual's confidence in her/his ability to prevent and to manage headaches (French et al., 2000). It has excellent internal consistency (Cronbach alpha of 0.9), and construct validity as demonstrated via significant correlations with related scales such as headache-related disability and psychological distress. The Headache-Specific Locus of Control (**HSLC**) consists of 33 items assessing the degree to which individuals believe that the variables controlling headache activity are primarily internal or external (Martin, Holroyd, & Penzien, 1990). It has three sub-scales (healthcare professional, internal, chance), all with good internal consistency (Cronbach alphas ranging from 0.84 to 0.88), and construct validity as demonstrated via significant correlations with related scales such as depression, disability, and catastrophizing.

Treatment Conditions

For each treatment, detailed therapist manuals and complementary participant handbooks were prepared, and are available from the authors. LCT and Avoid + CBT were administered weekly over eight weeks, and Avoidance was scheduled for weeks 1, 4 and 8. The relaxation training (progressive muscle relaxation) and cognitive therapy techniques used in two of the treatment conditions were based on the procedures employed in Martin, Forsyth and Reece (2007) derived from the book *Psychological Management of Chronic Headaches* (Martin, 1993). The treatment conditions were as follows.

Avoidance. This intervention involved education, identifying headache triggers, and managing headache triggers through avoidance, including developing Avoidance of Headache Trigger Plans.

Learning to Cope with Triggers ('LCT'). The therapist manual specified principles for identifying triggers and deciding what strategies to use for each trigger. 'Planned exposure' was presented as serving three potential functions:

- Exposure as an 'experiment' to see if the alleged trigger does indeed precipitate headaches. This approach would seem worth trying with triggers for which there is less support in the research literature for their capacity to precipitate headaches (e.g., foods - Hannington, 1967; Savi et al., 2002), and for which there is limited evidence for the individual (e.g., person believes a headache was precipitated by a particular 'trigger' and has avoided that 'trigger' ever since, hence not testing the 'hypothesis').
- Exposure to achieve desensitization/habituation/adaptation. This approach should be considered for sensory triggers such as flicker, glare, eyestrain and noise, for which our laboratory studies have shown prolonged exposure leads to an attenuated pain response (Martin, 2001; Martin et al., 2006; Martin, 2000). It is also the strategy of choice for anxiety as a trigger (e.g., Barlow, 2004).
- Exposure to enable practising coping skills. This strategy is often advocated in the stress management literature, whereby mild stress is induced using imaginal techniques, and coping skills are used to reduce stress levels (e.g., Meichenbaum, 1985).

The intervention advised participants that exposure should generally be seen as the strategy of choice for triggers such as stress and negative affect, and sensory triggers (e.g., visual disturbance, noise), while avoidance should generally be seen as the strategy of choice for triggers that are not consistent with a healthy lifestyle such as toxic smells, hunger, dehydration and lack of sleep. Menstruation was identified within this treatment condition as being a cue to focus on the other triggers that often give rise to headaches in combination with hormonal factors, with a view to desensitizing those other triggers or avoiding them at the critical time of the month.

The therapist endeavoured to ensure that parameters of exposure to triggers (length of exposure and intensity of trigger stimulus) were manipulated such that they fell short of precipitating significant headaches. Hence, discussions took place with the participants as to what length and intensity of trigger exposure would lead to a headache, and participants were encouraged to use exposure such that it would be insufficient to precipitate a headache. Participant efforts at exposure to triggers were reviewed in treatment sessions and feedback was given to shape up how they used exposure.

Evidence-based strategies were used whenever possible, such as for stress (e.g., Meichenbaum, 1985) and anxiety (e.g., Barlow, 2004). Exposure was used to 'real' and 'imagined' triggers, in the clinic and in the natural environment. Relaxation training (progressive muscle relaxation - learning a skill through instruction and practice, and then using the developed skill to achieve a relaxed state, with CD provided to participants), and cognitive therapy techniques (identifying and challenging dysfunctional thoughts), were used to facilitate exposure. Procedures from the 'return of fear' literature were used to prevent a trigger that had been desensitized from regaining the capacity to precipitate headaches (Lang, Craske, & Bjork, 1999). Relapse prevention strategies were included. LCT began with education and proceeded by developing Headache Action Plans for exposure or avoidance. The therapist manual included 22 appendices on how to manage different triggers including example Headache Action Plans for each one.

Avoidance combined with CBT ('Avoid + CBT'). This intervention involved the same content as the Avoidance condition, with the remaining time devoted to relaxation training and cognitive therapy techniques.

Waiting-List ('Waitlist'). Participants received no treatment until after an assessment equivalent to the post-treatment assessment of participants in the other three conditions.

Therapists and Treatment Integrity

The therapists were two doctoral-trained psychologists. Their training in the study protocols began with extensive reading and an 8-hour workshop led by the primary author who has been involved in the treatment of headache as a clinician, researcher and educator for 40 years. Weekly meetings were held between the therapists, study coordinator and the primary author throughout the period of the study. The therapists followed the manuals and discussed their cases during these weekly meetings, and supervision was provided.

Experts on treatment fidelity (e.g., Bellg et al., 2004; Davidson et al., 2003; Moncher & Prinz, 1991) recommend a number of procedures for promoting treatment fidelity including using treatment manuals and regular supervision of the treatment agent, as described above. The additional method used was to video sessions and have a therapist blind to treatment allocation determine which type of treatment (LCT or Avoid + CBT) the participant received for randomly selected videos. This procedure was followed with 44 videos and identification was correct for all but one video (97.8% correct).

Treatment adherence was promoted by the use of participants self-monitoring homework tasks (Davidson et al., 2003). The therapists checked these records at each session and when compliance was not 100%, reasons for noncompliance were investigated and steps taken to improve adherence.

Procedure

Pre-treatment assessment and training. Individuals volunteering for the study were screened by telephone on the selection criteria (except for diagnosis), and individuals meeting these criteria were invited to a pre-treatment assessment session at which informed consent procedures were completed. An assessment followed that included headache diagnosis using a structured interview based on the criteria in the International Classification of Headache Disorders, 2nd edition (Headache Classification Subcommittee of IHS, 2004). The diagnostic interview included assessment of medication overuse, and a primary diagnosis of Probable medication-overuse headache resulted in exclusion from the study.

Participation ceased at this point for all individuals not meeting all selection criteria. These individuals were provided with information pertaining to referral sources. For eligible individuals, demographic information was recorded and an assessment of headache triggers. Participants then completed the HTAQ, HMSES and HSLC. The latter part of the session was used for training in self-monitoring headaches and medication use. All assessment and treatment sessions were carried out at Monash Medical Centre in Melbourne, Australia.

Baseline self-monitoring. Participants commenced self-monitoring the day after the pre-treatment assessment session and continued for four weeks prior to treatment, in accordance with the Guidelines for Trials of Behavioral Treatments for Recurrent Headache (Penzien et al., 2005).

Randomization. During the 4-week self-monitoring period, participants were randomly allocated to the four groups. The Investigator with statistical responsibility and no involvement with participants (John Reece), took responsibility for the randomization. A computer-generated sequence was used, and CONSORT guidelines followed.

Treatment. Treatment followed over the next eight weeks.

Post-treatment assessment. Participants were asked to continue self-monitoring headaches and medication consumption for four weeks after treatment concluded. In addition, they were asked to complete the HTAQ, HMSES and HSLC. At the completion of this period, participants in the Waitlist condition were offered LCT.

Four and 12-month follow-up assessments. Four months after completing treatment, participants were asked to resume self-monitoring headaches and medication consumption for a 4-week period. They were requested to complete the HTAQ, HMSES and HSLC. This was repeated 12 months after completing treatment.

Results

Data Preparation and Analysis Strategy

Headache intensity ratings from the diaries were averaged across the waking day (all ratings including ratings of zero were added and divided by the number of ratings recorded) to produce a composite index ('mean daily headache rating'). The Guidelines for Trials of Behavioral Treatments for Recurrent Headache (Penzien et al., 2005) state "Investigators are urged to report a measure of headache activity (preferably headache frequency) as their principal dependent measure" (p. S125). This study has used 'mean daily headache rating' as the primary outcome measure (referred to in the Guidelines as 'headache activity/index') rather than headache frequency, as this measure is based on all three parameters of headache – frequency, duration and intensity. This avoids potential problems such as treatment leading to less frequent but longer lasting and more intense headaches being judged as effective.

Medication scores (pill counts) from the diaries were added for each day ('mean daily medication use') as recommended by the International Headache Society (IHS Committee on Clinical Trials in Tension-type Headache, 2000). Individual values for these scores along with scores for all of the other measures described above, were entered for each participant at each of the four experimental phases into a single SPSS 20 spreadsheet.

Analysis of the main outcome variables was based around analysis of covariance (ANCOVA), which is the preferred approach to the analysis of randomized controlled trials, providing assumptions are met (Vickers & Altman, 2008). Primary analyses focussed on the post-treatment scores. Secondary analyses (described below) incorporated the two sets of follow-up data.

For outcome variables with sub-scales, an initial single-factor between-subjects multivariate analysis of covariance (MANCOVA) was conducted by combining the sub-scales from each main outcome measure. The single between-subjects factor for all analyses was made up of the four experimental groups. Baseline scores formed the covariates, with post-treatment scores as the dependent outcomes. These MANCOVAs were followed by single-factor between-subjects univariate ANCOVAs on each outcome variable. Significant

univariate ANCOVAs were followed by post-hoc pairwise comparisons of the covariate-adjusted post-treatment group means with Sidak-adjusted α values.

Clinical significance was assessed by calculating the pre- to post-treatment percentage reduction in mean daily headache ratings for each participant. Participants were assessed to have demonstrated a clinically significant reduction if they exhibited greater than 50% reduction in headaches. χ^2 contingency table analysis was used to assess the relationship between treatment and the number of participants who met the clinical significance criterion. Number Needed to Treat analyses for the three active treatment groups were based on these figures.

Analyses of these two main outcome measures also considered the potential moderating impact of primary headache diagnosis (i.e., migraine versus tension-type headache) on treatment outcome. Pre- to post-treatment change scores on mean daily headache ratings and mean daily medication consumption were calculated and then analyzed using 2 x 4 between-subjects factorial ANOVAs with the two headache diagnosis groups and the four treatment conditions forming the two factors.

The data for those participants who formed the waiting-list control group and who then chose to receive the coping treatment once the main protocol had ceased were analyzed using Hotelling's T^2 (for those outcome measures with multiple sub-scales) followed by univariate single-factor within-subjects ANOVAs. For this single group, only pre- and post-treatment data were available.

Secondary analyses incorporating the two sets of follow-up data were conducted via 3 x 4 mixed (i.e., split-plot) factorial analyses of variance (ANOVA). The single between-subjects factor was made up of three treatment groups (follow-up data were not collected for the waitlist condition, so this was excluded from these analyses); the single within-subjects factor was made up of the four experimental phases (i.e., baseline, post-treatment, four month follow-up, 12 month follow-up). Again, initial multivariate analyses of outcome

variables with sub-scales were conducted, followed by univariate analyses of separate outcome measures.

A manipulation check was assessed by analysing the data from the Trigger Specific Avoidance Scale of the HTAQ. Pre- to post-treatment change scores were calculated, and a single-factor between-subjects ANOVA based on the four treatment conditions was used to evaluate differences in the treatment effect on trigger avoidance.

For all inferential tests, an effect size is reported (either Cohen's *d* or η_p^2) with a 95% confidence interval for the effect size.

Assumption Testing

All outcome variables were assessed for normality and homogeneity of variance. All variables exhibited some degree of non-normality as assessed by visual inspection of histograms, stem-and-leaf and normality plots, and normality tests, but the degree of non-normality was not considered serious enough to warrant transformation, the use of non-parametric procedures, or the use of descriptives other than *M* and *SD*, particularly given the sample size and the robustness of the chosen procedures to deviations from normality (Norman, 2010).

Heterogeneity of variance was evident for several variables. In each instance, a power transformation based on the power figure recommended by SPSS 20 resulted in the assumption being met. For the analyses presented below, descriptive statistics are reported using the original metric and all inferential tests are based on the transformed data.

Multiple imputation for missing data was considered, but given the amount of missing data, particularly for the follow-up phases, and its non-random nature (as assessed by both MCAR testing and visual inspection of missing data patterns), it was felt that any form of missing value estimation was unwarranted, including Last Observation Carried Forward (LOCF). To affirm this decision, simulated data sets based on both LOCF and multiply imputed data were analyzed, resulting in no notable difference in the overall pattern of

findings. Participant attrition was varied with only one participant lost from the waitlist control group for both headache ratings and medication use, to a pre – post attrition of 25% in the LCT group. Full participant numbers are provided in Table 2. As mentioned above, there were no systematic or significant associations between treatment completion and any demographic or outcome variables.

Response to Treatment as a Function of Headache Diagnosis

To investigate whether headache diagnosis was related to treatment response, the five diagnostic groups were aggregated in two ways. First, the three migraine groups were combined and compared with the two TTH groups, and second, the two chronic groups were removed to yield a comparison between episodic migraine (migraine with and without aura) and episodic TTH. For the first comparison, there was no evidence that the main treatment effects on 'mean daily headache rating' or 'mean daily medication consumption' were moderated by diagnosis. A 2 x 4 between-subjects factorial MANOVA was conducted on the combined headache and medication use pre- to post-treatment change scores with diagnosis (migraine, TTH) and treatment group (four treatment conditions) forming the two factors. No significant multivariate interaction was found, $\Lambda = .94$, $F(2, 83) = 0.88$, $p = .51$, $\eta_p^2 = .03$, 95% CI [< .01, .06]; nor was there a significant multivariate main effect for headache diagnosis, $\Lambda = .98$, $F(2, 83) = 0.75$, $p = .48$, $\eta_p^2 = .02$, 95% CI [< .01, .09].

For the second comparison, the same analytical design was used. No significant multivariate interaction, $\Lambda = .88$, $F(2, 68) = 2.00$, $p = .15$, $\eta_p^2 = .13$, 95% CI [.04, .20], or headache diagnosis main effect, $\Lambda = .97$, $F(2, 68) = 0.45$, $p = .65$, $\eta_p^2 = .03$, 95% CI [< .01, .07] was evident.

Impact of Treatment on Trigger Avoidance

All four groups recorded less avoidance of triggers on the HTAQ after treatment compared to before treatment: Waitlist, -16.1%; Avoidance, -13.1%; Avoid + CBT, -11.0%; and LCT, -26.8%. These results were confirmed by inspection of the post-treatment means

for trigger avoidance after controlling for baseline variance, which revealed the LCT group (M = 29.03) to have a notably lower association with trigger avoidance by comparison with the Waitlist (M = 34.72), Avoidance (M = 37.15), or Avoid + CBT (M = 38.52) groups. Inferential testing on both of these sets of data failed to find a significant difference among the four treatment conditions.

Analysis of Primary Outcome Variables from Pre- to Post-treatment

Table 2 provides the descriptive statistics and univariate ANCOVA results for the two primary outcome variables of 'mean daily headache rating' and 'mean daily medication consumption', and the cognitive variables of self-efficacy and locus of control. Inspection of the Table reveals the following changes from pre- to post-treatment on the two primary outcome variables, with a plus sign indicating an increase and a minus sign indicating a decrease, that is, improvement: (i) headache rating: Waitlist, +11.0%; Avoidance, -13.2%; Avoid + CBT, -30.0%; and LCT, -35.9%; and (ii) medication consumption: Waitlist, +15.4%; Avoidance, -9.0%; Avoid + CBT, -19.4%; and LCT, -27.9%.

A single-factor between-subjects MANCOVA combining the two primary outcome variables revealed a significant multivariate effect for treatment, $\Lambda = .78$, $F(6, 178) = 3.90$, $p = .001$, $\eta_p^2 = .12$, 95% CI [.02, .18]. As reported in Table 2, both of the subsequent univariate ANCOVAs were significant. For headache rating, post hoc testing revealed a significant difference between the Waitlist and two treatment conditions: Avoid + CBT, $p = .006$, $d = 1.00$, 95% CI [0.37, 1.60], and LCT, $p = .002$, $d = 1.11$, 95% CI [0.49, 1.73]. For medication use, the only significant post hoc result was between the Waitlist and LCT conditions, $p = .001$, $d = 1.14$, 95% CI [0.52, 1.76]. Figure 2 shows the pattern of baseline to post-intervention change for the four treatment conditions for these two main outcome variables.

Clinical significance was assessed using the widely accepted criterion of a 50% or greater reduction in headaches from pre- to post-treatment ('treatment responders'). A 4 x 2 χ^2 contingency table analysis revealed a significant association between treatment condition

and percentage of treatment responders, χ^2 (3, N = 97) = 12.50, 95% CI [1.16, 27.70], p = .006, V = .36. The LCT group revealed the highest percentage of treatment responders (46%), followed by Avoidance (42%), Avoid + CBT (29%), and Waitlist (4%). Post hoc testing of the significant contingency table χ^2 was based on the examination of standardized residuals (i.e., the standardized difference between the observed cell count and the count expected under the null hypothesis), which revealed that the low number of participants in the Waitlist and the high number of participants in the LCT group who exhibited clinically significant change made a notable contribution to the significant χ^2 result.

The fact that 42% of the Avoidance group were treatment responders was unexpected not only in terms of the rationale of the study, but also in view of the fact that the mean headache rating of this group decreased by only 13.2%. Consequently some analyses were carried out to explore differences between the 10 participants who achieved clinically significant improvement and the 14 participants who did not, in the Avoidance group. Specifically, *t*-tests were carried out on age, chronicity, and change from pre- to post-treatment in trigger avoidance (HTAQ), while χ^2 was used for gender and diagnosis. The findings for age, chronicity, trigger avoidance and gender were all non-significant, but the χ^2 for diagnosis was significant, χ^2 (1, N = 24) = 5.71, 95% CI [1.14, 18.92], p = .05, V = .49. This finding reflected the fact that only 5 out of 18 migraineurs were classified as treatment responders, whereas 5 out of 6 TTH sufferers were so classified.

The same clinical significance figures were used to generate a Number Needed to Treat (NNT) Analysis for the three treatment groups. For both LCT and Avoidance, NNT = 3 (95% CI for LCT [1.6 – 4.9]; 95% CI for Avoidance [1.7 – 6.1]). NNT = 4 for the Avoid + CBT group, 95% CI [2.2 – 18.4].

Sixteen participants in the Waitlist group received LCT after completing their second assessment. Pre- and post-treatment data were analyzed for these participants for the primary

outcome variables. Both headache rating and medication consumption decreased over treatment (headaches: pre-treatment, $M = 0.78$, $SD = 0.34$; post-treatment, $M = 0.42$, $SD = 0.28$; medication: pre-treatment, $M = 2.61$, $SD = 2.50$; post-treatment, $M = 2.26$, $SD = 2.18$). These represent decreases in headaches and medication consumption of 46.2% and 13.4%, respectively. A Hotelling's T^2 comparing pre- to post-treatment change on these combined measures found a significant multivariate reduction, $T^2 = 1.87$, $F(2, 14) = 13.12$, $p = .001$, $\eta_p^2 = .65$, 95% CI [.22, .78]. A significant univariate reduction was found for headaches, $F(1, 15) = 27.62$, $p < .001$, $\eta_p^2 = .65$, 95% CI [.27, .79] but not for medication consumption, $F(1, 15) = 4.16$, $p = .06$, $\eta_p^2 = .22$, 95% CI [< .01, .50], although the trend for medication consumption was in the expected direction.

Analysis of Secondary Outcome Variables from Pre- to Post-treatment

With respect to the cognitive variables, a significant multivariate treatment effect was found for the three combined locus of control scales, $\Lambda = .70$, $F(9, 214.32) = 3.70$, $p < .001$, $\eta_p^2 = .11$, 95% CI [.03, .19], with the internal factors and chance sub-scales revealing significant univariate treatment effects. For the internal factors sub-scale, post-hoc testing revealed significant differences between the Waitlist group and all three treatment groups: Avoidance, $p = .009$, $d = 0.94$, 95% CI [0.34, 1.53], Avoid + CBT, $p = .001$, $d = 1.18$, 95% CI [0.56, 1.79], and LCT, $p < .001$, $d = 1.22$, 95% CI [0.60, 1.83]. These findings reflect that the internal locus of control scores decreased from pre- to post-treatment for the Waitlist group but increased for the three treatment groups with the LCT group showing the largest increase (see Table 2). For the chance sub-scale the only significant post-hoc result was between the Waitlist and LCT conditions, $p = .02$, $d = 0.88$, 95% CI [0.29, 1.47]. This finding reflected that the chance locus of control scores did not change for the Waitlist group from pre- to post-treatment, but decreased in the LCT group (see Table 2). No significant treatment effect was found on the HMSES, although the largest change (i.e., increased self-efficacy) was for the LCT group.

Analyses of Outcome Variables at 4- and 12-month follow-up

A 3 x 4 multivariate split-plot factorial MANOVA combining the two primary outcome measures (headache rating and medication consumption) was conducted, with treatment condition (three treatment groups) and time (pre, post, 4-month follow-up and 12-month follow-up) forming the two factors. This analysis failed to find a significant multivariate phase by treatment group interaction, $\Lambda = .70$, $F(12, 102) = 1.68$, $p = .083$, $\eta_p^2 = .17$, 95% CI [< .01, .20], although a significant multivariate phase main effect was evident, $\Lambda = .57$, $F(6, 51) = 6.55$, $p < .001$, $\eta_p^2 = .44$, 95% CI [.16, .54]. Despite the absence of a multivariate interaction, a significant univariate phase by treatment group interaction was found for medication consumption (see Table 2). Examination of the simple main effects associated with this result found that the LCT group varied significantly across the four phases, $\Lambda = .79$, $F(3, 54) = 4.77$, $p = .005$, $\eta_p^2 = .21$, 95% CI [.02, .18], in particular the change from baseline to post-treatment, $p = .004$, $d = 0.76$, 95% CI [0.28, 1.23], and baseline to 4-month follow-up, $p = .041$, $d = 0.60$, 95% CI [0.14, 1.05].

For the three combined sub-scales of the HSLC, no significant phase by treatment group interaction was found, $\Lambda = .72$, $F(18, 110) = 1.07$, $p = .39$, $\eta_p^2 = .15$, 95% CI [< .01, .18], but there was a significant phase main effect, $\Lambda = .38$, $F(9, 55) = 9.91$, $p < .001$, $\eta_p^2 = .62$, 95% CI [.37, .68]. At the univariate level, no significant interactions were found, but two of the three sub-scales revealed a significant univariate phase main effect. No significant phase by treatment group interaction was found for the HMSES, although a significant phase main effect was evident.

Discussion

Treatment and Trigger Avoidance

One condition did not try to change trigger avoidance (Waitlist), two conditions aimed to increase avoidance (Avoidance and Avoid + CBT), and one condition encouraged avoidance of some triggers and exposure to other triggers (LCT). Contrary to these objectives, all four

conditions were associated with a decrease in avoidance of triggers. The decrease in avoidance was over twice as large in the LCT condition as the two avoidance conditions, but differences did not reach statistical significance.

There are a number of different responses to these findings. The failure of the two avoidance conditions to increase avoidance of triggers could be taken as evidence of a problem with advice to avoid triggers, namely that it is difficult to avoid triggers or too high a cost to avoid triggers. Alternatively, perhaps participants already avoided triggers and hence more avoidance was not possible. It could be argued that for advice to avoid triggers to be given a proper test of treatment efficacy, then a way needs to be found for achieving compliance with avoidance instructions.

As the four groups did not significantly differ in terms of changes in trigger avoidance, but some differences were found in terms of improvement in headaches and medication consumption, it could be suggested that trigger avoidance is not very important in terms of headache management. Such a conclusion would seem premature, however, as the decrease in trigger avoidance was twice as large in the LCT group as the two avoidance groups.

Efficacy of Advice to Avoid all Headache Triggers

The Avoidance condition was associated with small decreases in headaches (13.2%) and medication consumption (9.0%) but differences between the Avoidance condition and Waitlist condition failed to reach significance on either variable. Surprisingly in view of these null findings, 42% of the participants in the Avoidance condition were treatment responders compared to 4% in the Waitlist condition. A higher proportion of TTH sufferers than migraineurs were treatment responders in the Avoidance condition.

Efficacy of Learning to Cope with Triggers

The findings for LCT were promising. LCT came out ahead of the other conditions on virtually all the measures used in the study and did significantly differ from the Waitlist condition in terms of changes on the two primary outcome measures from pre- to post-

treatment. Improvement in the LCT group on headaches and medication was approximately three fold the improvement in the Avoidance group. The positive results for LCT were further supported by the favorable outcomes obtained by participants who started in the Waitlist condition and then subsequently received LCT. LCT also included the highest percentage of treatment responders at 46%. These results were achieved in a group of participants who had suffered from headaches for an average of 24 years. The results at follow-up were less impressive than the results at post-treatment but the LCT group showed a greater reduction in levels of medication consumption, from pre-treatment to 4-month follow-up than the other treatment groups.

The LCT condition was associated with more improvement than the Avoid + CBT condition on all variables, but there were a limited number of statistically significant findings related to a comparison between these conditions. The LCT group significantly differed from the Waitlist group, but this was not the case for the Avoid + CBT group, on the following variables: treatment responder rate (percentage achieving a 50% reduction in headaches); medication consumption; and the chance sub-scale of the locus of control scale. Possible explanatory factors as to why more significant differences did not emerge include the power of CBT as a treatment for headaches (Rains et al., 2005), and the overlap between LCT and Avoidance as LCT supports avoidance of some triggers. On the other hand, even meagre evidence that doing exactly the reverse of conventional wisdom with some triggers (i.e., encouraging exposure rather than avoidance) seems to result in better rather than worse outcomes, is an important finding.

A meta-analytic review that summarized the results for behavioral treatment of migraine and TTH concluded that average improvement ranged from 33% to 55% (Rains et al., 2005). Hence, the headache reductions associated with LCT of 35.9% (LCT condition) and 46.2% (participants in the Waitlist condition who subsequently received LCT) fall into this range. This is encouraging as LCT is an approach to trigger management rather than a

comprehensive approach to headache treatment. Combining LCT with other treatment techniques should lead to enhanced treatment efficacy.

The LCT condition significantly differed from the Waitlist condition in terms of pre- to post-treatment changes on the internal and the chance sub-scales of the locus of control scale, and although falling short of statistical significance, the largest change in self-efficacy occurred in the LCT group. These cognitive changes should be advanageous to participants in the LCT condition (Marlow, 1988).

Trial Limitations

The main limitation of the trial was that although the sample was quite large for a trial of behavioral treatment of headaches, a larger sample would have given more statistical power. The sample included individuals suffering from migraine and TTH, and included individuals with different subtypes of headaches, but statistical analyses did not reveal any significant differences or even trends in terms of a differential treatment response as a function of headache diagnosis, with the exception of the treatment responder rate in the Avoidance condition. Most studies of behavioral headache treatment do not include both migraine and TTH in their samples but where they have, differential treatment effects tend not to emerge (e.g., Martin, Forsyth, & Reece, 2007; Martin, Nathan, Milech et al., 1989).

Given the method of recruiting participants, the results are generalizable to members of the community who self-identify themselves as having headaches that need treatment. The characteristics of the sample in terms of gender, age, diagnosis and chronicity, were very similar to samples we have recruited for other behavioral treatment trials (e.g., Martin et al., 2007). The mean chronicity of 26.3 years emphasises that this was group of sufferers who had had headaches for a very long time.

Future Research

Future research should look at developing LCT in a number of ways. First, as it is only an approach to trigger management, it would be appropriate to evaluate how best to combine

LCT with other approaches to treatment, to yield the most effective comprehensive approach to the management of recurrent headaches. The other treatment approaches could be behavioral, for example, the techniques used in CBT, or pharmacological. Second, given the desire of both busy physicians and patients for brief interventions, it would be of value to determine whether a more abbreviated version of LCT can be developed that yields benefits similar to those produced by the present 8-session LCT program. Third, in order to improve long-term outcomes for LCT, it would be useful to examine how relapse prevention training could be improved or booster sessions might best be employed to sustain initial treatment gains. Finally, research is needed into treatment mechanisms and predictors of treatment success for LCT, both to guide the enhancement of its efficacy and to permit its selective delivery to those most likely to benefit from this approach.

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References

Andrasik, F., Lipchik, G. L., McCrory, D. C., & Whitrock, D. A. (2005). Outcome measurement in behavioral research: Headache parameters and psychosocial outcome. *Headache, 45*, 429-437.

Andress-Rothrock, D., King, W., & Rothrock, J. (2010). An analysis of migraine triggers in a clinic-based population. *Headache, 50*, 1366-1370.

Asmundson, G. J. G., Norton, P. J., & Vlaeyen, J. W. S. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 3-24). Oxford, UK: Oxford University Press.

Barlow, D. H. (2004). *Anxiety and its disorders* (2nd ed.). New York: Guilford Press.

Bellg, A. J., Borrelli, B., Resnick, B., Hecht, J., Minicucci, D. S….Czajkowski, S. (2004). Enhancing treatment fidelity in health behavior change studies: Best practices and recommendations from the NIH Behavior Change Consortium. *Health Psychology, 23*, 443-451.

Blanchard, E. B., Andrasik, F., Neff, D. F., Jurish, S. E., & O'Keefe, D. M. (1981). Social validation of the headache diary. *Behavior Therapy, 12*, 711-715.

Blau, J. N., & Thavapalan, M. (1988). Preventing migraine: A study of precipitating factors. *Headache, 28*, 481-483.

Bussone, G. (2004). Pathophysiology of migraine. *Neurological Sciences*. 25:s239.

Chabriat, H., Danchot, J., Michel, P., Joire, J. E., & Henry, P. (1999). Precipitating factors of headaches. *Headache, 39*, 335-338.

Collins, F. L., & Thompson, J. K. (1979). Reliability and standardization in the assessment of self-reported head pain. *Journal of Behavioral Assessment, 1*, 73-86.

Crombez, G., Eccleston, C., Van Damme, S., Vlaeyen, J. W. S., & Karoly, P. (2012). Fear-avoidance model of chronic pain: The next generation. *Clinical Journal of Pain, 28,* 475-483.

Davidson, K. W., Goldstein, M., Kaplan, R. M., Kaufmann, P. G., Knatterud, G. L.…Whitlock, E. P. (2003). Evidence-based behavioral medicine: What is it and how do we achieve it? *Annals of Behavioral Medicine, 26,* 161-171.

Deniz, O., Aygul, R., Kocak, N., Orhan, A., & Kaya, M. D. (2004). Precipitating factors of migraine attacks in patients with migraine with and without aura. *The Pain Clinic, 16,* 451-456.

Eysenck, H. J. (1979). The conditioning model of neurosis. *Behavioral and Brain Sciences, 2,* 155-199.

French, D. J., Holroyd, K. A., Pinell, C., Malinoski, P. T., O'Donnell, F., & Hill, K. R. (2000). Perceived self-efficacy headache-related disability. *Headache, 40,* 647-656.

Friedman, D. I., & De Ver Dye, T. (2009). Migraine and the environment. *Headache, 49,* 941-952.

Hannington, E. (1967). Preliminary report on tyramine headache. *British Medical Journal, 1,* 550-551.

Hayes, S. C., Strosahl, K., Wilson, K. G., Bissett, R. T., Pistorello, J…McCurry, S. M. (2004). Measuring experiential avoidance: A preliminary test of a working model. *The Psychological Record, 54,* 553-579.

Headache Classification Subcommittee of the International Headache Society (2004). The International Classification of Headache Disorders, 2nd ed. *Cephalalgia, Supplement 1, 24,* 1-151.

Ierusalimschy, R., & Moreira Filho, P. F. (2002). Precipitating factors of migraine attacks in patients with migraine without aura. *Arq Neuropsiquiatra, 60,* 609-613.

International Headache Society Committee on Clinical Trials in Tension-type Headache (2000). *Members' Handbook 2000*. Oslo, Norway: Scandinavian University Press.

Karli, N., Zarifoglu, M., Calisir, N., & Akgoz, S. (2005). Comparison of pre-headache phases and trigger factors of migraine and episodic tension-type headache: Do they share similar clinical pathophysiology? *Cephalalgia, 25*, 444-451.

Kelman, L. (2007). Triggers or precipitants of the acute migraine attack. *Cephalalgia, 27*, 394-402.

Lang, A. J., Craske, M. G., & Bjork, R. A. (1999). Implications of a new theory of disuse for the treatment of emotional disorders. *Clinical Psychology: Science and Practice, 6*, 80-93.

Leeuw, M., Goossens, M. E. J. B., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. S. (2007). The fear-avoidance model of musculoskeletal pain: A current state of scientific evidence. *Journal of Behavioral Medicine, 30*, 77-94.

Leone, M., Vila, C., & McGowan, C. (2010). Influence of trigger factors on the efficacy of almotriptan as early intervention for the treatment of acute migraine in a primary care setting: the START study. *Expert Review of Neurotherapeutics, 10*, 1399-1408.

Marcus, D. (2003). Chronic headache. *Headache and Pain, 14*, 139-144.

Marlowe, N. (1998). Self-efficacy moderates the impact of stressful events on headache. *Headache, 38*, 662-667.

Martin, N. J., Holroyd, K. A., & Penzien, D. H. (1990). The Headache-Specific Locus of Control Scale: Adaptation to recurrent headaches. *Headache, 30,* 729-734.

Martin, P. R. (1993). *Psychological management of chronic headaches*. New York: Guilford Press.

Martin, P. R. (2000). Headache triggers: to avoid or not to avoid, that is the question. *Psychology and Health, 15*, 801-809.

Martin, P. R. (2001). How do trigger factors acquire the capacity to precipitate headaches? *Behaviour Research and Therapy, 39,* 545-554.

Martin, P. R. (2010a). Managing headache triggers: Think 'coping' not 'avoidance'. *Cephalalgia, 30,* 634-637.

Martin, P. R. (2010b). Behavioral management of migraine headache triggers: Learning to cope with triggers. *Current Pain and Headache Reports, 14,* 221-227.

Martin, P. R., Forsyth, M. R., & Reece, J. (2007). Cognitive-behavioral therapy versus temporal pulse amplitude biofeedback training for recurrent headache. *Behavior Therapy, 38,* 350-363.

Martin, P. R., Lae, L., & Reece, J. (2007). Stress as a trigger for headaches: Relationship between exposure and sensitivity. *Anxiety, Stress and Coping, 20,* 393-407.

Martin, P. R., & MacLeod, C. (2009). Behavioral management of headache triggers: Avoidance of triggers is an inadequate strategy. *Clinical Psychology Review, 29,* 483-495.

Martin, P.R., Nathan, P.R., Milech, D., & van Keppel, M. (1989). Cognitive therapy versus self-management training in the treatment of chronic headache. *British Journal of Clinical Psychology, 28,* 347-361.

Martin, P. R., Reece, J., & Fordyce, M. (2006). Noise as a trigger for headaches: Relationship between exposure and sensitivity. *Headache, 46,* 962-972.

Meichenbaum, D. (1985). *Stress inoculation training*. New York: Pergamon Press.

Moncher, F. J., & Prinz, R. J. (1991). Treatment fidelity in outcome studies. *Clinical Psychology Review, 11,* 247-266.

Moses, E. B., & Barlow, D. H. (2006). A new unified treatment approach for emotional disorders based on emotion science. *Current Directions in Psychological Science, 15,* 146-150.

Nash, J. M. D. M. W., Williams, D. M., Nicholson, R., Trask, P. C. (2006). The contribution of pain-related anxiety to disability from headache. *Journal of Behavioral Medicine, 29*, 61-67.

Norman, G. (2010). Likert scales, levels of measurement, and the 'laws' of statistics. *Advances in Health Sciences Education*, *15*, 625–632.

Penzien, D. B., Andrasik, F., Freidenberg, B. M., Houle, T. T., Lake, A. E….Wittrock, D. A. (2005). Guidelines for trials of behavioral treatments for recurrent headache, first edition: American Headache Society Behavioral Clinical Trials Workgroup. *Headache, 45(Suppl 2),* S110-S132.

Peters, M., Abu-Saad, H. H., Robbins, I., Vydelingum, V., Dowson, A., & Murphy, M. (2005). Patient's management of migraine and chronic daily headache: A study of members of the Migraine Action Association (United Kingdom). *Headache, 45,* 571-581.

Philips, H. C., & Hunter, M. (1981). Pain behavior in headache sufferers. *Behavior Analysis and Modification, 4*, 257-266.

Rains, J. C., Penzien, D. B., McCrory, D. C., & Gray, R. N. (2005). Behavioral headache treatment: History, review of the empirical literature, and methodological critique. *Headache, 45 (S2),* S92-S109.

Savi, L., Rainero, I., Valfre, W., Gentile, S., Lo Giudice, R., & Pinessi, L. (2002). Food and headache attacks. *Panminerva Medica, 44*, 27-31.

Scharff, L., Turk, D. C., & Marcus, D. A. (1995). Triggers of headache episodes and coping responses of headache diagnostic groups. *Headache, 35,* 397-403.

Skaer, T. L. (1996). Clinical presentation and treatment of migraine. *Clinical Therapeutics, 18,* 229-245.

Snyder, C. R., & Pulvers, K. M. (2001). Dr Seuss, the coping machine. In C. R. Snyder (Ed.), *Coping with stress: Effective people and processes*. New York, NY: Oxford University Press.

Spierings, L. H., Ranke, A. H., & Honkoop, P. C. (2001). Precipitating and aggravating factors of migraine versus tension-type headache. *Headache, 41*, 554-558.

Suls, J., & Fletcher, B. (1985). The relative efficacy of avoidant and nonavoidant coping strategies: A meta-analysis. *Health Psychology, 4*, 249-288.

Vickers, A. J., & Altman, D. G. (2001). Statistics notes: Analysing controlled trials with baseline and follow up measurements. *British Medical Journal, 323,* 1123-1124.

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain, 85*, 317-332.

Wood, A., & Martin, P. R. (in preparation). Development of the Headache Triggers Avoidance Questionnaire.

World Health Organization (2006). *Neurological disorders*. Switzerland: WHO.

Table 1

Demographic and Headache Characteristics of Sample

Variable	Treatment Group				
	Waitlist	Avoidance	Avoid + CBT	LCT	Total
Gender					
Male	5	11	15	12	43
Female	27	18	19	20	84
Age in years					
M	46.91	48.28	48.94	44.53	47.08
SD	15.15	12.57	13.65	13.85	13.91
Education					
Less than Yr 12	4	1	3	5	13
Yr 12	6	4	7	4	21
Cert/Diploma	9	12	10	9	40
Undergrad deg	6	9	11	11	37
Postgrad deg	7	3	3	3	16
Occupation					
Managers	3	3	5	4	15
Professionals	8	9	11	5	33
Tech & trades	0	1	0	2	3
C & PSW	6	2	3	6	17
Clerical & admin	4	2	0	5	11
SW, MO & D, L	0	0	1	2	3
Not employed	5	4	4	4	17
Retired	4	6	8	4	22
Student	2	2	2	0	6
Headache diagnosis					
MwA	5	10	7	5	27
MwoA	15	9	8	15	47
C**M**	2	2	3	2	9
FE**TTH**	7	5	9	5	26
C**TTH**	3	3	7	5	18
Headache chronicity in years					
M	27.34	27.63	27.40	23.69	26.26
SD	16.42	13.36	14.29	13.09	14.27

Yr 12 – Year 12 (twelfth grade or senior year in North America)
Cert/Diploma – post secondary education below level of undergraduate degree
C & PSW – Community and Personal Service Workers
SW, MO & D, L – Sales workers, Machinery Operators & Drivers, Labourers
MwA - **Migraine** with Aura
MwoA - **Migraine** without Aura
C**M** - Chronic **Migraine**
FE**TTH** - Frequent Episodic **Tension–Type Headache**
C**TTH** - Chronic **Tension-Type Headache**

Table 2

Descriptive Statistics and Analysis of Covariance Results for Experimental Groups on Outcome Measures

	Treatment Group											ANCOVA				
	Waitlist			Avoidance			Avoid + CBT			LCT						
Outcome Measure and Phase	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*F*	*df*	*p*	η_p^2	95% CI η_p^2
Mean daily headache rating													5.81	3, 92	.001	.16	.03, .27
Baseline	26	0.91	0.93	29	0.91	0.72	32	1.10	0.73	30	0.92	0.82					
Post-Treatment	25	1.01	0.92	24	0.79	0.95	24	0.77	0.71	24	0.59	0.55					
4 Month				18	0.62	0.85	25	0.73	0.69	24	0.68	0.80					
12 Month				22	0.70	0.90	21	0.69	0.61	22	0.66	0.53					
Mean daily medication use													5.02	3, 92	.003	.14	.02, .25
Baseline	25	2.72	2.96	28	3.23	3.38	32	2.83	3.30	30	3.15	2.69					
Post-Treatment	24	3.14	3.33	24	2.94	3.20	24	2.28	2.53	24	2.27	2.30					
4 Month				18	3.10	3.62	25	2.23	2.11	24	2.41	2.36					
12 Month				22	3.03	3.53	21	1.92	1.57	22	2.83	2.43					
HMSES													1.03	3, 94	.38	.03	< .01, .10
Baseline	32	108.25	18.88	29	108.45	21.91	34	106.32	26.23	32	101.53	21.89					
Post-Treatment	25	115.80	19.15	24	113.42	21.75	26	126.08	22.31	24	127.71	26.00					
4 Month				22	117.73	21.31	25	130.92	27.51	24	123.38	28.01					
12 Month				22	117.91	23.58	23	128.65	30.32	22	127.91	26.08					
HSLC – Healthcare Professional													1.33	3, 94	.27	.04	< .01, .12
Baseline	32	29.00	6.25	29	26.45	9.28	34	27.79	8.80	32	27.00	7.05					
Post-Treatment	25	28.00	6.00	24	27.08	8.78	26	27.92	6.66	24	24.46	6.57					
4 Month				22	26.73	8.39	25	27.24	7.45	24	24.33	7.38					
12 Month				22	27.05	7.57	23	25.13	8.15	22	23.50	7.82					
HSLC – Internal													7.76	3, 94	<.001	.20	.06, .32
Baseline	32	38.94	10.88	29	36.17	9.19	34	36.76	11.55	32	34.81	7.13					
Post-Treatment	25	36.36	8.34	24	39.38	9.13	26	42.19	6.03	24	41.71	7.48					
4 Month				22	38.82	8.15	25	42.28	8.78	24	38.46	8.19					
12 Month				22	39.14	5.15	23	41.09	9.43	22	36.77	9.77					
HSLC – Chance													3.33	3, 94	.02	.10	< .01, .10
Baseline	32	30.38	8.12	29	33.34	7.71	34	32.62	10.26	32	33.59	7.86					
Post-Treatment	25	30.04	6.76	24	29.79	10.04	26	27.81	10.18	24	25.83	8.90					
4 Month				22	28.91	9.42	25	27.28	10.22	24	26.54	10.32					
12 Month				22	28.50	9.36	23	27.30	10.65	22	23.77	10.20					

Note. ANCOVA results are the results of a single-factor between-subjects ANCOVA on post-treatment means adjusted for baseline variance.

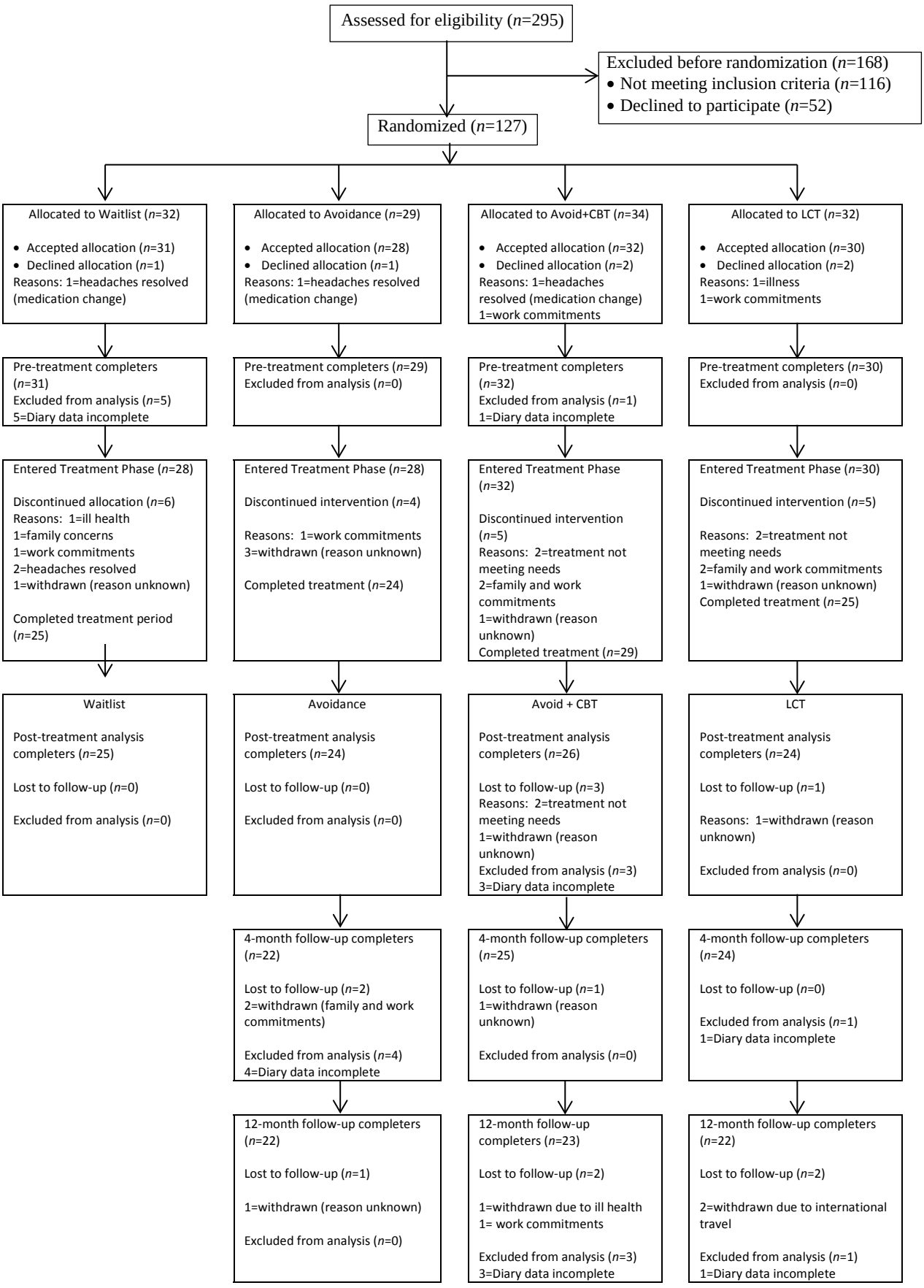

Figure 1. Flow chart of participant recruitment and involvement in study.

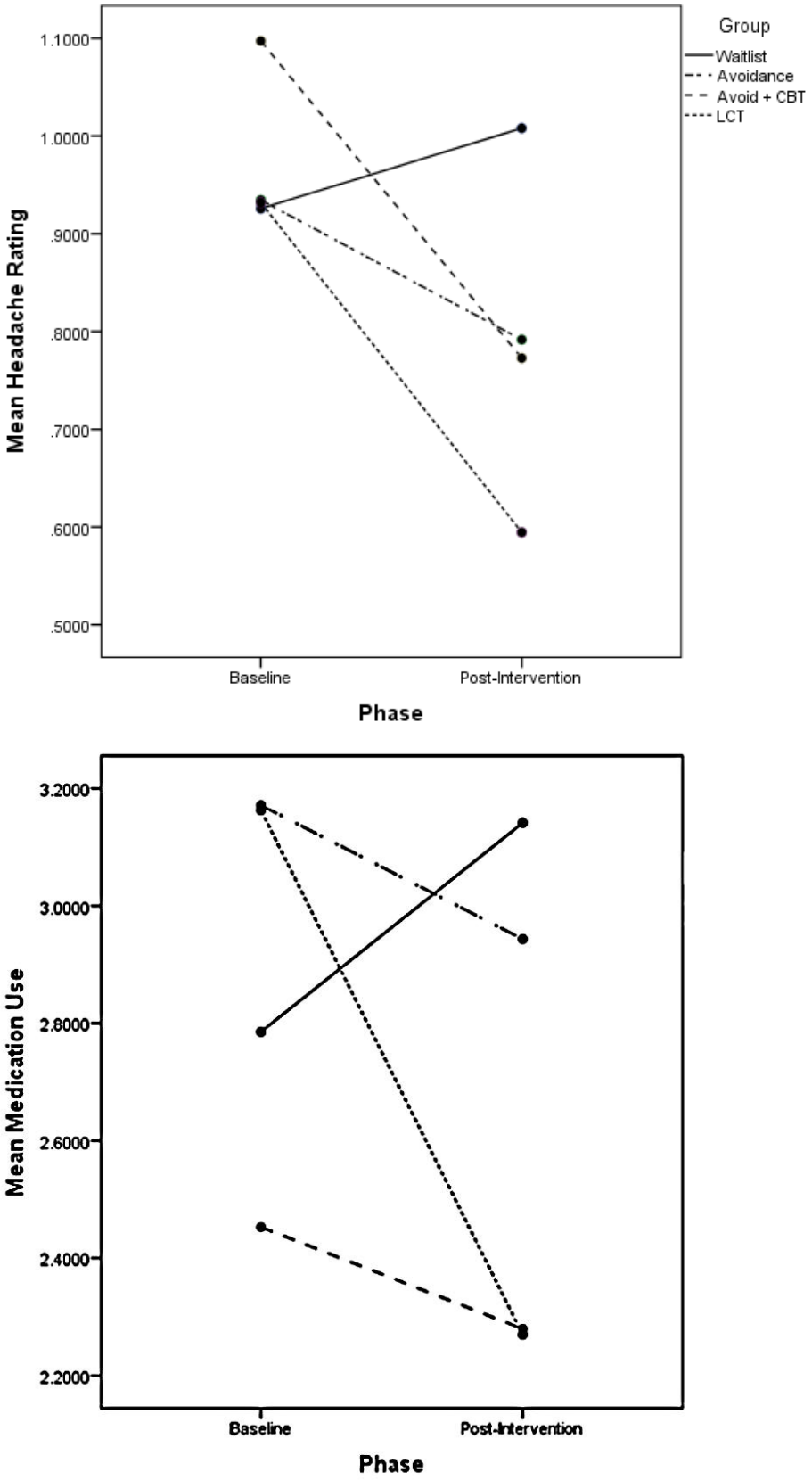

Figure 2. Group by phase interactions for the two primary outcome variables.