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Research Submission

Noise as a Trigger for Headaches: Relationship Between Exposure and Sensitivity

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Objective.—This study investigated how triggers acquire the capacity to precipitate headaches.

Background.—Traditional clinical advice is that the best way to prevent headache/migraine is to avoid the triggers. Avoidance of anxiety-eliciting stimuli, however, results in sensitization to the stimuli, so is there a danger that avoidance of migraine/headache triggers results in decreased tolerance for the triggers?

Design.—One hundred and fifty subjects, 60 of whom suffered from regular headaches, were randomly assigned to 5 experimental conditions, defined by length of exposure to the headache trigger of noise.

Methods.—Subjects attended a laboratory session divided into 3 phases: preintervention test, intervention (1 of 5 levels of exposure to the trigger), and postintervention test. Response to the intervention was measured in terms of noise tolerance, sensitivity to noise, and nociceptive response to noise.

Results.—A curvilinear relationship was found between length of exposure to the trigger and pain response for individuals who do not suffer from regular headaches, that is, short exposure was associated with sensitization and prolonged exposure with desensitization. The relationship for headache patients was less clear.

Conclusions.—The findings are consistent with the proposition that 1 etiological pathway to suffering from frequent headaches is via trying to avoid, or escape from, potential trigger factors. These results suggest that the traditional clinical advice to headache patients, that the best way to prevent migraine/headache is to avoid the triggers, runs the risk of establishing an insidious sensitization process thereby increasing headache frequency.

Key words: tension-type headache, migraine, triggers, sensitization, desensitization, etiology

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A functional model of chronic headache has been proposed in which headaches are conceptualized in terms of their controlling variables.[1,2] The model aims to understand the variance in headaches by considering the antecedents and consequences of headaches. The immediate antecedents of headaches are the stimuli that precipitate or aggravate headaches, that is, the trigger factors. Martin et al[2] demonstrated that the most commonly reported triggers are stress, anxiety, glare, and noise. This study also found that triggers could be grouped into a small number of independent, interpretable patterns including "negative affect" (stress, anxiety, anger, and depression) and "visual disturbance" (flicker, glare, and eyestrain). While most of the literature on headache triggers are based on self-reports, some triggers have been experimentally validated including negative affect,[3,4] visual disturbance,[4] hunger,[3] and noise.[5] Much attention has been given to the issue of whether migraine and tension-type headache have the same or different

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triggers, and the majority of studies have failed to find differences,[6-8] but some have.[9]

Traditional medical advice to headache patients is to avoid triggers. Schulman and Silberstein[10] counsel, for example, that "wherever possible, practitioners should help susceptible patients learn to avoid triggers," and Skaer[11] states that "migraine prevention is best achieved by avoidance of known migraine triggers." The logic of this advice is clear, but is it realistic, and is there a danger that avoidance of a trigger may increase sensitivity to it? It is well established that avoidance of stimuli that give rise to anxiety results in increased sensitivity or sensitization to the anxiety-eliciting stimuli or situations.[12] Anxiety is treated by prolonged exposure to anxiety-eliciting situations as this results in a desensitization process whereby the situations lose their capacity for eliciting anxiety.

There is very limited empirical support for the clinical practice of encouraging avoidance of headache triggers. Blau and Thavapalan[13] encouraged migraineurs to avoid all precipitating factors and reported a reduction of 50% in attack frequency in 19 out of 23 patients. However, a number of methodological flaws in the study limit its utility. For example, all patients were given advice on "how to abort attacks by quickly taking an antinauseant and analgesic tablets" (p. 481) so that the effects of this advice were confounded with the effects of the advice to avoid precipitants. Also, the 50% reduction was based on a comparison between a retrospective estimate of attack frequency in the 3 months before consultation with "noting attacks" during the 2 months after consultation, so that changes in frequency were confounded with different approaches to measurement. The study did not include any control conditions.

In contrast to the findings of Blau and Thavapalan,[13] a study by Philips and Jahanshahi[14] provided some empirical support for the potential of exposure to reduce sensitivity to headache triggers. Philips and Jahanshahi investigated the effects on chronic headache patients of exposure to the headache trigger of noise. They found that exposure was associated with reduced pain response whereas avoidance of exposure led to increasing intolerance.

Whether avoidance or exposure is more appropriate in the management of headache disorders depends on the process by which sensitivity to trigger factors is acquired. Most triggers are stressful, aversive, uncomfortable, irritating, disturbing, or unpleasant in some way, and most probably have the capacity, when presented at high intensity or for long duration, to elicit headaches in most people. Nevertheless, both between- and within-subject variations in responsivity to these trigger factors are apparent, that is, some individuals are more vulnerable to developing headaches in response to these factors than others, and vulnerability can change across time for individuals. Development of a chronic headache disorder can be conceptualized as becoming sensitized to various headache trigger factors such that these factors precipitate headaches more readily.

How does this sensitization process come about? Two opposing theoretical accounts seem plausible. One position suggests that on each occasion a trigger is associated with a headache, the link between the 2 is strengthened or consolidated, as occurs through learning by practice or repetition (repetition theory). An alternative position is that observation by a headache patient of a link between a trigger and a headache (whether the observation is accurate or inaccurate) results in escape/avoidance behavior whereby the patient seeks to minimize exposure to the trigger. This avoidance of triggers sensitizes the individual to the trigger in the same way that avoidance of anxiety-inducing stimuli results in increased anxiety to future presentations of the stimuli (avoidance theory). The anxiety literature shows that short exposure to anxiety-provoking stimuli results in increments to subsequent anxiety responses to the stimuli, while prolonged exposure to anxiety-provoking stimuli results in decrements to subsequent anxiety responses to the stimuli.[15] Short exposure underlies the maintenance of phobias as phobics try and escape or avoid threatening stimuli, thus minimizing exposure, whereas long exposure is the basis for the highly effective treatments for anxiety such as various forms of desensitization, flooding, and implosion.[12] The repetition account suggests that the critical process in sensitization is simply the pairing of the triggers with headaches, whereas the avoidance account suggests that the critical process is the maladaptive reaction of the patient to the triggers. Not surprisingly, the research literature indicates that headache patients do respond to headaches via avoidance of the trigger factors.[16,17]

We completed a study in which participants were exposed to the experimentally validated headache trigger factor of visual disturbance for 1 of 5 different durations.[18] Both the repetition and avoidance theories predict that short exposure to a trigger factor would lead to increased sensitivity to the trigger factor. However, with respect to long exposure, the repetition theory predicts further increases in sensitivity whereas the avoidance theory predicts decreased sensitivity. The results of the study strongly supported the avoidance theory.

In another study, 6 chronic headache patients repeatedly attended the laboratory for sessions involving exposure to visual disturbance.[19] The results demonstrated that repeated, prolonged exposure to this headache trigger led to desensitization with participants experiencing less visual disturbance, less negative affect, and less head pain in response to the trigger.

There is no reason to believe that all factors that can precipitate headaches have acquired the capacity via the same process. Research findings do suggest that different trigger factors precipitate headaches via different peripheral physiological mechanisms. For example, Martin and Teoh[4] found that headaches triggered by visual disturbance were associated with increased blood pressure, whereas Martin et al[5] reported that headaches triggered by noise were associated with elevated temporal pulse amplitude.

Reported here are the results of a study that replicated Martin[14] except noise was used rather than visual disturbance. Noise was selected for this study because it has been listed as the fourth most common trigger of headache[2] and has been experimentally validated.[5] Also, it lends itself to experimentally testing theories about the relationship between length of exposure to a trigger and sensitivity to a trigger.

The objective of the study was to test the competing predictions from the 2 theories. On the basis of our earlier research with visual disturbance, it was hypothesized that for both individuals who suffered from regular headaches and those who did not suffer from regular headaches, short exposure to noise would result in increased sensitivity and prolonged exposure would result in decreased sensitivity.

SUBJECTS AND METHODS

Subjects.—Subjects were recruited from students and staff of the University of New England via advertisements on the campus net and notice boards. The advertisements emphasized that the researchers not only particularly wished to recruit subjects who suffered from regular headaches, but also wished to recruit subjects who did not suffer from regular headaches, so that the responses of the 2 groups could be compared.

One hundred and fifteen subjects completed the study, of whom 42 (34%) were male. Eighty-one (70%) were students and 34 (30%) were staff. The age distribution was as follows: 18 to 24 years, n = 63; 25 to 34 years, n = 17; 35 to 44 years, n = 18; 45 to 54 years, n = 12; and 55+ years, n = 4. To compare individuals who suffered from frequent headaches with those who did not, an operational definition of "regular headache patient" was employed that defined "regular" as experiencing "on average, at least 1 headache per fortnight." On this basis, the sample included 60 (52%) patients who suffered from regular headaches, and 55 (48%) who did not suffer from regular headaches, subsequently referred to as "headache patients" and "non-headache patients," respectively.

A psychologist diagnosed the headaches according to the criteria of the Headache Classification System of the International Headache Society.[20] The following diagnoses were established: "migraine without aura," n = 6; "migraine with typical aura," n = 8; "episodic tension-type headache," n = 24; "chronic tension-type headache," n = 3; and "headache of the tension-type not fulfilling above criteria," n = 11. Diagnoses could not be established for 8 subjects due to missing data. The periods over which subjects had suffered from regular headaches were as follows: "less than 6 months," n = 2; "6 to 12 months," n = 5; "1 to 3 years," n = 9; "3 to 10 years," n = 19; and "more than 10 years," n = 24. There were missing data for 1 subject.

Subjects were randomly assigned to the 5 experimental conditions, defined by duration of exposure to noise, resulting in 22 to 24 subjects in each condition.

This consisted of 12 "headache patients" in each of the 5 conditions, and 11 "non-headache patients" in each of 3 conditions ("none," "long," and "very long"). The other 2 conditions had 10 ("short") and 12 "non-headache patients" ("very short") in them.

This research was carried out in accordance with the guidelines of the National Health and Medical Research Council, and was approved by the Human Research Ethics Committee of the University of New England. Written informed consent was obtained from all subjects. No incentives were offered for participation.

Design.—Sessions in the study were divided into 3 phases. The first phase consisted of a preintervention test that involved exposure to a noise stimulus. Subjects were instructed to indicate the point in time when they could no longer tolerate the noise stimulus. The noise was then turned off. The noise was turned off after 5 minutes if subjects had not indicated reaching their point of intolerance previously. Measures of experience of noise and headache intensity in response to the noise stimulus were taken 15 seconds after the onset of the stimulus and at termination of the stimulus. The second phase was the intervention, which comprised 1 of 5 durations of exposure to the noise stimulus (none, very short, short, long, and very long). The final phase consisted of the postintervention test, which was a repeat of the preintervention test, to assess the effects of the intervention.

Tests and Intervention.—The noise stimulus was 85 dB of white noise, chosen for 3 reasons. The first was that it would score highly on the scale of noise annoyance designed by Kryter,[21] as it was composed of multiple frequencies of sound (white noise), of high intensity (85dB), and resembled a commonly encountered stimulus with negative valence (ie, an un-tuned television set). The second reason was that it compared in nature and intensity to noise stimuli used in past experimental research but of longer duration, as recommended.[22] Finally, the composition and nature of the stimulus posed no threat to the auditory system of participants over the duration of exposure.[23]

The intervention phase of the study was divided into 140 15-second trials separated by 5-second intertrial intervals. In the anxiety literature, "short exposure" equates to around 5 to 10 minutes. Hence, for the *very short exposure* condition, the noise stimulus was presented in 20 of the 140 trials for a total exposure duration of 5 minutes. In the anxiety literature, the principle governing length of exposure is to continue exposure until anxiety begins to subside in the anxiety-eliciting situation, which usually translates in practice to exposure in excess of 30 minutes.[24] Hence, for the *very long exposure* condition, the noise was turned on in all 140 trials for a total exposure duration of 35 minutes. In a study of the effects of repeated, prolonged exposure to visual disturbance, we found that 100 trials for a total exposure of 25 minutes, produced desensitization,[19] so the *long exposure* condition was set at this level. The *short exposure* condition was positioned midway between the very short exposure and the long exposure conditions at 60 trials for 15 minutes of exposure. In the *no exposure* condition, the noise stimulus was not presented in any of the trials. Hence, the 5 exposure conditions used in the intervention, expressed in minutes, were 0, 5, 15, 25, and 35. When noise was presented in some trials and not in others, the noise trials were randomly distributed across the session. It should be noted that exposure was not continuous, as occurs in the anxiety literature.

Measures.—The following measures were recorded during the pre- and postintervention tests. Tolerance of noise was measured by recording how long subjects could tolerate the noise stimulus presented continuously. The maximum time of exposure was set at 5 minutes. Sensitivity to noise was measured by requesting subjects to rate the degree of aversion to noise experienced 15 seconds after the onset of the noise stimulus, and again at offset of the noise stimulus. The rating scale used ranged from 0 ("not at all present") to 5 ("very severely present"). Nociceptive response to noise was measured by requesting subjects to rate head pain intensity prior to the onset of the noise stimulus, 15 seconds after onset, and at offset of the noise stimulus. The rating scale used ranged from 0 ("no headache") to 5 ("an intense incapacitating headache"). The time line for these measures is shown as Figure 1.

Procedure.—Subjects were asked to attend the laboratory on a day when they were headache-free. If subjects had a headache at the arranged time, a new appointment was scheduled. The session began

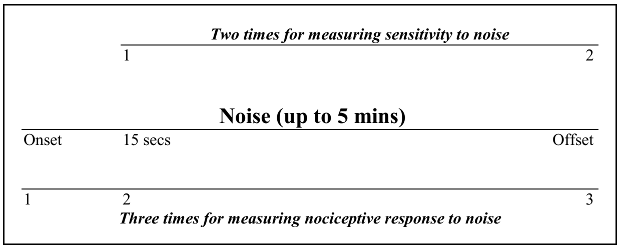

Fig 1.—Time line for measures.

with subjects completing a consent form followed by a brief questionnaire that included questions pertaining to demographic information and headaches. Instructions pertaining to the session were then given to subjects via a tape recorder, and subjects were provided with the opportunity to ask questions. Next, subjects completed the preintervention test. A 5-minute break followed during which subjects were asked to relax before subjects completed the 140 trials that constituted the intervention. Another 5-minute break followed the intervention before subjects completed the postintervention test. The session concluded with subjects completing a brief questionnaire inquiring about their experiences during the session. The total duration of the session was approximately 1 hour.

RESULTS

Preliminary Analyses.—To calibrate the noise stimulus, subjects were asked "how much noise did you experience in the session compared to the highest level of noise that you encounter on a weekly basis?" Twenty-eight percent responded "less" or "much less," 30% responded "about the same," and 43% responded "more" or "much more."

Headaches were reported in the session by 56 subjects (49%). Most of the headaches were low intensity with only 12 subjects using ratings of 3 ("a painful headache, but one during which you could continue at your job") or higher. Of those who reported experiencing a headache during the session, 81% indicated that the headache was "similar" or the "same" as their usual type of headaches.

Fifty-nine subjects (51%) tolerated the noise stimulus for the maximum period of 5 minutes in the preintervention test. A Mann-Whitney *U*-test found a significant difference in the noise tolerance time for the headache patients versus the non-headache patients, $U = 1185.00$, $Z = 2.80$, $P = .005$, with non-headache patients (Mean = 234,000 ms) demonstrating higher tolerance times than headache patients (Mean = 173,000 ms).

Dependent Variables Used in Main Analyses.—It was decided to select or create 7 dependent variables from the measures recorded for analyzing the effect of the intervention as this would result in a participant to variable ratio of approximately 16 to 1, which generally results in satisfactory power and is considered a satisfactory ratio for obtaining a reliable estimate of the pattern of covariation and replicable results from multivariate analyses.[25] One measure of tolerance for noise following the intervention was selected: the maximum time that noise could be tolerated in the postintervention test (T1). Two measures of sensitivity to noise following the intervention were selected: the ratings of noise measured 15 seconds after onset (S1), and immediately after offset (S2), of the noise in the postintervention test. Four measures of nociceptive response to noise following the intervention were created as follows: (a) ratings of headache intensity measured immediately prior to the postintervention test minus ratings of headache intensity measured immediately prior to the preintervention test (ie, ratings at postintervention correcting for differences preintervention) (H1); (b) ratings of headache intensity measured 15 seconds after onset of the noise in the postintervention test minus ratings of headache intensity measured immediately prior to the preintervention test (H2); (c) ratings of headache intensity measured immediately after offset of the noise in the postintervention test minus ratings of headache intensity measured immediately prior to the preintervention test (H3); and (d) ratings of headache intensity measured immediately after offset of the noise in the postintervention test minus ratings of headache intensity measured immediately after offset of the noise in the preintervention test (H4). These variables were equivalent to the ones used in the study of visual disturbance as a trigger factor,[14] therefore facilitating comparisons between the studies.

The 7 variables were evaluated in terms of whether they met the assumptions underlying multivariate analysis of variance. S1 and S2 did, H2 and H4 were skewed, and H1 and H3 were severely skewed. Consequently, a square root transformation was applied to H2 and H4, and a cube root transformation was applied to H1 and H3. Tests for both multivariate and univariate homogeneity of variance on the transformed variables did not reveal significant violations of these assumptions. The distribution of scores for T1 was more problematic, exhibiting severe skew, a ceiling effect, and lack of variability. Transformation was not effective in improving the shape of the distribution for the T1 scores; consequently, these data were not included in the parametric analyses reported below, and instead were analyzed separately using nonparametric procedures.

Main Analyses.—Six of the 7 variables (H1, H2, H3, H4, S1, and S2) were entered in a 2 × 5 factorial between-subjects multivariate analysis of variance (ANOVA). The between-subjects factors were "headache" (2 levels—headache patient and non-headache patient) and "exposure condition" (the 5 exposure durations). The multivariate interaction between headache and exposure condition was not significant, $\Lambda = .90$, $F(24, 350.07) = 0.98$, $P = .50$, partial $\eta^2 = .06$ (<.01, .05) nor was the multivariate main effect for exposure condition, $\Lambda = .80$, $F(24, 350.07) = 0.95$, $P = .54$, partial $\eta^2 = .05$ (< .01, .05). Note, the figures in parentheses following the effect size measure (η^2) are the upper and lower bounds of the 95% confidence intervals around η^2. The multivariate main effect for headache was significant, $\Lambda = .79$, $F(6, 100) = 4.45$, $P < .001$, partial $\eta^2 = .21$ (.05, .30). Follow-up univariate ANOVAs on the 6 dependent variables revealed a significant difference between the 2 headache groups for all variables, with effect sizes ranging from partial $\eta^2 = .04$ for H4 to partial $\eta^2 = .15$ for H2: H1, $F(1, 105) = 15.35$, $P < .001$, partial $\eta^2 = .13$ (.03, .25); H2, $F(1, 105) = 18.40$, $P < .001$, partial $\eta^2 = .15$ (.04, .27); H3, $F(1, 105) = 14.82$, $P < .001$, partial $\eta^2 = .12$ (.03, .24); H4, $F(1, 105) = 4.55$, $P = .035$, partial $\eta^2 = .04$ (< .01, .13); S1, $F(1, 105) = 6.38$, $P = .013$, partial $\eta^2 = .06$ (< .01, .16); S2, $F(1, 105) = 8.18$, $P = .005$, partial $\eta^2 = .07$ (.01, .18).

On the 4 measures of nociceptive response to noise, the headache group consistently used higher pain ratings, and on the 2 measures of sensitivity to noise, the headache group consistently rated higher degree of aversion to noise. Given the lack of any significant interaction between headache and exposure condition, the subsequent analyses were performed separately for the headache group and the non-headache group.

The repetition and avoidance theories predict different relationships between length of exposure to a trigger factor and headache response to the trigger factor. If headache response to the trigger factor is plotted against length of exposure to the trigger factor, then support for the repetition theory would be evidenced by an approximation to a straight line from lower left to upper right, whereas support for the avoidance theory would be evidenced by an approximation to an inverted U-curve. These hypothetical relationships are shown in Figure 2. This translates statistically to a linear relationship between exposure and headache response supporting the repetition theory, in contrast to a quadratic relationship between exposure and headache response supporting the avoidance theory.

To test these alternative predictions, analyses of orthogonal polynomials (ie, trend analyses) were carried out on each variable for the 2 groups separately, and the results revealed different trends for the 2 headache groups. For non-headache patients, 4 of the 6 dependent outcomes revealed significant quadratic trends: H1, $F(1, 50) = 6.53$, $P = .013$, partial $\eta^2 = .12$ (<.01, .29); H2, $F(1, 50) = 6.43$, $P = .014$, partial $\eta^2 = .11$ (<.01, .28); H3, $F(1, 50) = 5.15$, $P = .025$, partial

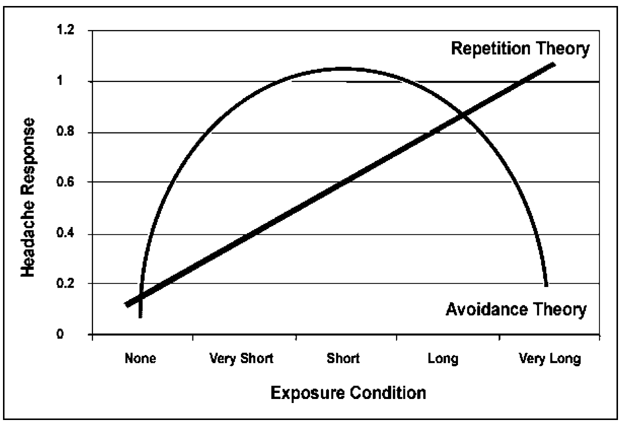

Fig 2.—Predicted results on the basis of 2 competing theories of how trigger factors acquire the capacity to precipitate headaches.

$\eta^2 = .09$ (<.01, .26); and H4, $F(1, 50) = 5.13$, $P = .028$, partial $\eta^2 = .09$ (<.01, .26). No other significant results were found for this group. It should be noted that all of the significant trend results were found on the 4 variables measuring nociceptive response to noise.

For headache patients, only 1 clearly significant trend was noted—a significant linear trend for H2, $F(1, 55) = 6.58$, $P = .013$, partial $\eta^2 = .11$ (<.01, .27)—although it is worth noting that the quadratic trend for H2 was trending toward significance, $F(1, 55) = 3.59$, $P = .063$, partial $\eta^2 = .05$ (<.01, .21), as was the linear trend for H1, $F(1, 55) = 3.88$, $P = .051$, partial $\eta^2 = .07$ (<.01, .22).

The trends for the 2 headache groups are shown in Figure 3.

For the T1 data, a single-factor between-subjects Kruskal-Wallis nonparametric ANOVA found a significant difference in scores across the 5 exposure conditions for non-headache patients, χ^2 (4, N = 55) = 11.60, $P = .021$, but not for headache patients, χ^2 (4, N = 60) = 2.30, $P = .68$. Post hoc testing on the significant non-headache patient result using Mann-Whitney

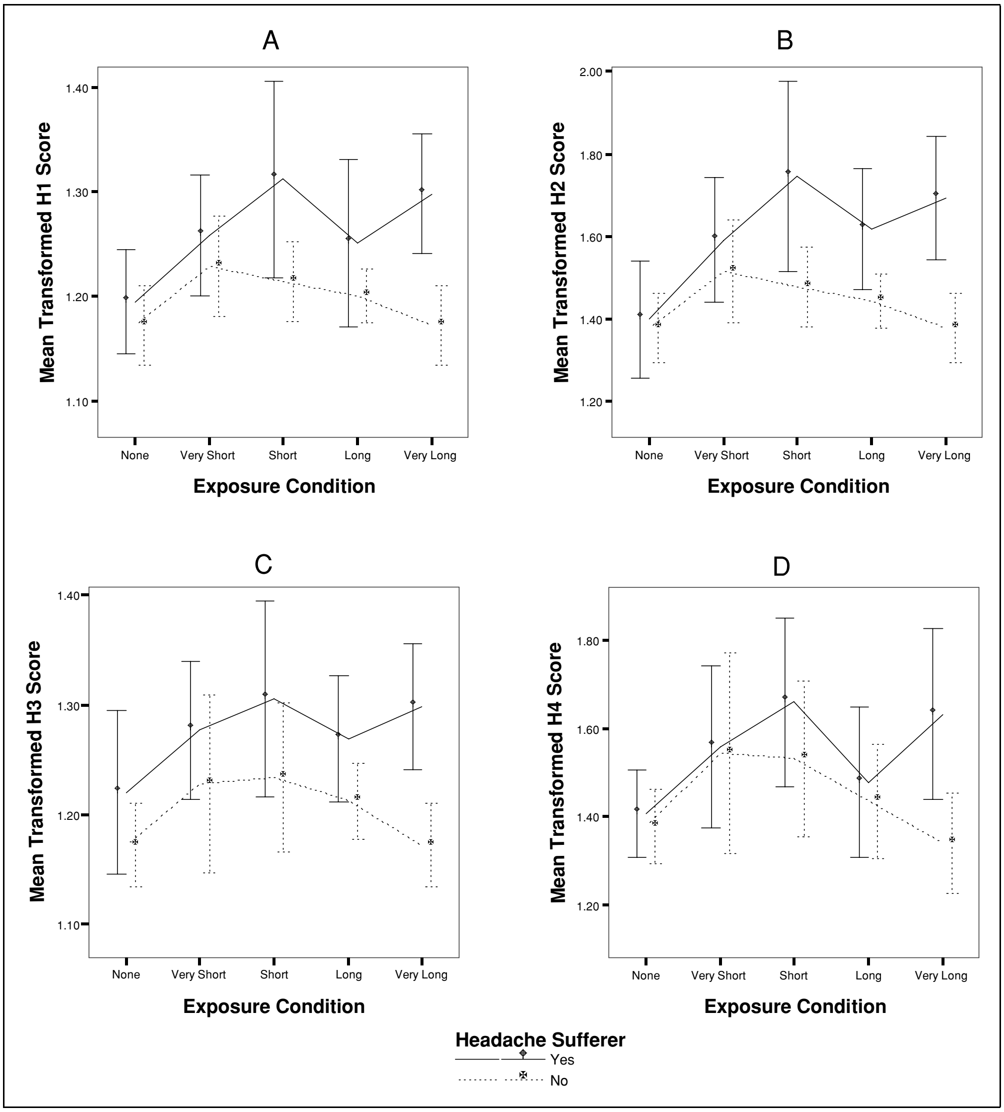

Fig 3.—Relationship between exposure condition and headache patient status for 4 major outcome variables. Bars show 95% confidence intervals.

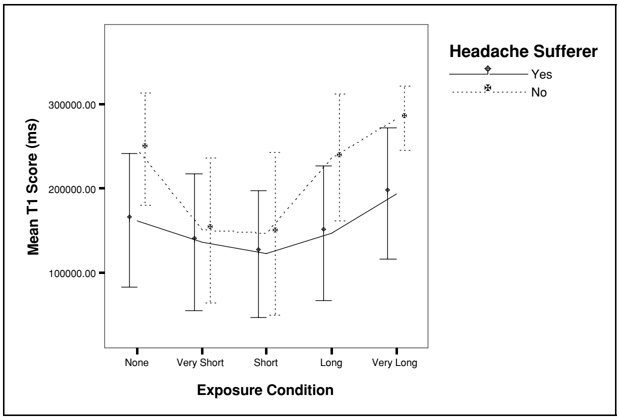

Fig 4.—Noise tolerance times (T1) (in milliseconds) for the 2 headache groups across 5 exposure conditions at postintervention. Bars show 95% confidence intervals.

U tests with a Bonferroni adjusted α level of .005 found no significant pairwise comparisons.

Further, a series of Mann-Whitney *U*-tests comparing the headache groups for each exposure condition found a significant difference between the two headache groups for the "very long" exposure condition, $U = 38.00$, $Z = 2.12$, $P = .034$, while the difference for the "long" exposure condition approached significance, $U = 38.00$, $Z = 1.86$, $P = .063$. In each case, the headache group showed less tolerance. These results are displayed in Figure 4.

The means and standard deviations of the variables shown in Figures 3 and 4 are presented in Table.

Table—Scores on 5 Outcome Measures Across Exposure Conditions for Headache Patients and Non-Headache Patients

	Exposure Condition				
Measure	None	Very Short	Short	Long	Very Long
Headache patients (n = 60)					
H1†					
Mean	1.19	1.25	1.31	1.25	1.29
SD	0.08	0.09	0.14	0.13	0.09
H2					
Mean	1.40	1.59	1.74	1.62	1.69
SD	0.22	0.24	0.36	0.23	0.23
H3					
Mean	1.22	1.28	1.31	1.27	1.30
SD	0.12	1.00	0.14	0.09	0.09
H4					
Mean	1.40	1.56	1.66	1.48	1.63
SD	0.16	0.29	0.30	0.27	0.31
T1‡					
Mean	162,153	136,473	122,436	146,473	193,960
SD	124,492	127,821	117,465	125,143	121,950
Non-headache patients (n = 55)					
H1					
Mean	1.17	1.23	1.21	1.20	1.17
SD	0.06	0.08	0.05	0.04	0.06
H2					
Mean	1.38	1.52	1.48	1.44	1.38
SD	0.12	0.20	0.13	0.10	0.12
H3					
Mean	1.17	1.23	1.23	1.21	1.17
SD	0.06	0.13	0.09	0.05	0.06
H4					
Mean	1.38	1.54	1.53	1.43	1.34
SD	0.12	0.36	0.25	0.19	0.17
T1					
Mean	246,552	150,454	146,256	236,164	282,949
SD	99,637	134,337	135,209	111,527	56,551

Note. See text for full description of measures.
†Scores for H1 to H4 are transformed scores on headache intensity ratings.
‡Scores for T1 are in milliseconds.

COMMENTS

Response to the question of how the noise stimulus compared to the noise experienced in every day life varied with some individuals reporting the noise was less than usual, some more than usual, and some the same as usual. The noise stimulus induced headaches in about half the sample, headaches that were usually of low intensity and of the same type as headaches experienced in everyday life.

The study was based on the assumption that chronic headaches can be conceptualized as individuals becoming sensitized to factors that can trigger headaches, and the findings of the study were consistent with this assumption. Hence, before the intervention, individuals who suffered from regular headaches had lower tolerance for the noise stimulus than individuals who did not suffer from regular headaches. Through the study, headache patients reported that they found the noise stimulus more aversive and it resulted in reports of more pain, than non-headache patients.

For individuals who do not suffer from regular headaches, the analyses strongly supported the avoidance theory as there were significant quadratic trends on all 4 measures of nociceptive response to noise. However, for individuals who do suffer from regular headaches, the results were less clear-cut. The only significant effect was a linear trend on 1 variable. There was a trend toward a linear effect on a second variable. These findings are in line with the repetition theory. However, there was a trend to a quadratic effect on 1 variable. Inspection of the figures showed a similar pattern of data for the headache and non-headache groups except for the "very long" exposure condition. In this condition, the non-headache group showed further desensitization beyond the "long" exposure condition whereas the headache group showed sensitization relative to the "long" exposure condition.

The results for the non-headache group paralleled the findings of Martin[18] using visual disturbance as a trigger factor, but the results for the headache group were different. The calibration process with the visual disturbance stimulus resulted in 78% responding that the stimulus was more or much more than usual, compared to 43% with the noise stimulus, so there is no suggestion in the data that the noise stimulus was a more extreme stimulus.

The data on noise tolerance following the intervention could not be analyzed using parametric statistics but inspection of the graphs in Figure 4 indicates a curvilinear relationship for both non-headache and headache groups, consistent with the avoidance theory. The graphs approximate U-curves rather than the inverted U-curves in the other figures, as tolerance and nociceptive response are inversely related.

The findings from individuals who do not suffer from regular headaches are consistent with the proposition that attempts to escape from or avoid potential headache triggers resulting in short exposure, may lead to a sensitization process that makes individuals more vulnerable to frequent headaches or even possibly developing a chronic headache disorder. The findings from individuals who suffer from regular headaches do not provide clear guidance as to whether avoidance or exposure to trigger factors is a better strategy from the perspective of desensitization/sensitization. The data hint at the possibility that for the trigger factor of noise, "long" exposure may be helpful but "very long" exposure may be unhelpful. This paper has argued for the potential benefits of exposure to triggers but it seems likely that exposure at too high a level will be counterproductive.

It should be noted that the terms "very short," "short," "long," and "very long," as used in this article, are somewhat arbitrary, as they are based on extrapolations from the anxiety literature. It is the relative nature of the exposure conditions that speaks to the 2 theories.

A limitation of the study is that the sample was recruited from students and staff of a university and hence the regular headache patients were not likely to be representative of the chronic headache population in the community. For example, young people were overrepresented in the sample, although age of participants did range to above 55.

The study did not investigate individual differences in response to exposure to the headache trigger of noise. Findings in the anxiety literature suggest this may be an important area for future research. For example, Beck and Shipherd have conducted a series of studies in which they exposed patients with panic disorder to the interoceptive cues that trigger panic attacks.[26-28] They have reported 2 distinct response patterns, 1 involving habituation and 1 involving sensitization.

The results of this study and the other studies on exposure to trigger factors raises the question of the biological mechanisms that might underlie sensitization and desensitization. Migraine is believed to be a neurovascular disorder, and central neuronal hyperexcitability is considered to be a key feature predisposing to migraine as it determines the threshold of response to trigger factors, and therefore susceptibility to attacks.[29,30] Could exposure to trigger factors affect central neuronal hyperexcitability? Central sensitization, comprising qualitative changes in the central processing of sensory information resulting in a lowering of pain thresholds, is considered to be a key mechanism in chronic tension-type headache.[31,32] Could exposure to trigger factors affect central sensitization? These questions await future research.

To place the findings on exposure to headache trigger factors in a broader context, it is worth noting that a debate is taking place as to whether avoidance or exposure to triggers is more appropriate in the management of allergic asthma. There is a long history of managing allergic asthma by allergen avoidance, but there is also a long history of treating allergic asthma by allergen immunotherapy, which involves graduated exposure to allergen trigger factors.[33] After recently reviewing the literature on allergen avoidance, 2 of the leading researchers in this field raised the question, "Are we going the wrong way?" and went on to conclude "Whether one should pursue the induction of tolerance by high-dose allergen exposure (either by injections, inhalations, or the oral route) as a treatment option for infants at high risk of allergic disease, with all the adherent risks, will be the challenge for the next decade."[34]

The findings from this study on noise build on the early work of Philips and Jahanshahi[14] and the 2 studies on visual disturbance,[18,19] and challenge the traditional clinical advice to avoid headache triggers. More research is needed, however, into the etiological question of how trigger factors acquire the capacity to precipitate headaches, and into the management issue of whether it is more appropriate to advise avoidance of trigger factors, or the alternative strategy of some form of controlled exposure designed to produce desensitization. Exposure to a point that falls just short of precipitating significant headaches seems worth exploring, with increases in the duration or intensity of exposure as the desensitization process unfolds, akin to allergen immunotherapy.

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