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**PR Martin****Abstract**

The standard clinical advice for individuals who suffer from recurrent headaches is that the best way to prevent headaches is to avoid the triggers. This editorial challenges that advice from a number of perspectives. First, there is little empirical support for such advice. Second, cognate literatures in the fields of chronic pain, stress and anxiety raise concerns about avoidance as a strategy. Third, studies have demonstrated that short exposure to a headache trigger results in increased sensitivity and prolonged exposure results in decreased sensitivity. Conclusions include that one aetiological pathway to developing a primary headache disorder may be via attempts to avoid triggers resulting in increased sensitivity to triggers. Also, clinicians need to become more flexible in the advice they give pertaining to triggers, namely they should think 'coping with triggers' rather than avoiding all triggers, as avoidance will sometimes be the preferred strategy, but often it will not be.

Keywords

Migraine, tension-type headache, triggers, avoidance, coping

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A central tenet of the management of recurrent headaches is that it is important to identify the headache triggers and then for the sufferer to avoid the triggers. This advice appears in authoritative medical texts and on the internet at sites ranging from the American Headache Society to Wikipedia. The logic of the advice is quite clear, and if all triggers could be avoided then no headaches should occur. Anyone with experience in treating headaches would recognize that it is 'easier said than done' as it is not always easy to identify triggers, triggers are diverse and omnipresent, some triggers cannot be avoided (e.g. menstruation), and trying to avoid all triggers can itself be stressful and lead to a restricted lifestyle.

There are additional problems that are less obvious, however. The advice to avoid triggers is given in a conceptual vacuum because we do not understand why triggers precipitate headaches. Is the capacity to precipitate a headache an inherent property of the trigger, or is it an acquired capacity, or some combination of the two, perhaps depending on the particular trigger? If exposure history to triggers is a factor in the acquisition and/or extinction of the capacity of the trigger to precipitate headaches, then the advice to avoid triggers could influence the capacity of the trigger to precipitate headaches (i.e. affect trigger potency). Put simply, does avoidance of triggers reduce tolerance for triggers and

thereby run the risk of increasing headaches in the longer term?

The author has recently completed a review of the literature relevant to the above questions (1) and the main arguments will be elaborated briefly here. If there was strong empirical support for encouraging avoidance of triggers, then the questions raised above would be of theoretical interest only, but such support is limited to a small number of group studies and case reports, none of which included relevant control conditions and all of which were liable to a number of methodological criticisms. Blau and Thavapalan (2) probably provided the strongest evidence by encouraging migraineurs to avoid all precipitating factors and reporting a reduction of 50% in attack frequency in 19 out of 23 patients. However, these authors also advised their patients '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 study compared a retrospective estimate of attack frequency in the 3 months before consultation with 'noting attacks' during the 2 months after consultation, meaning that pre- vs. post-intervention differences

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could potentially be attributable to the differing measurement approaches employed on each occasion. The study did not assess treatment adherence and did not include long-term follow-up.

If the capacity to precipitate a headache were an inherent property of all triggers and not modifiable by exposure history to the triggers, then advice to avoid triggers should raise few concerns, but this position is not plausible. Genetic studies demonstrate that some people are born with a predisposition to develop a headache disorder and some people have a headache disorder for most of their lives. However, genetics explains only a proportion of the variance and no one is born a headache sufferer—headaches are an acquired disorder. Longitudinal studies show that headache disorders come and go across the life span. If the capacity of a trigger to precipitate a headache is an inherent and immutable property of the trigger then these data imply that presence of triggers comes and goes synchronously with the headache disorder, which seems unlikely.

Consideration of cognate literatures raises concerns about the advice to avoid headache triggers. In the chronic pain literature, fear-avoidance models have been developed, which contend that individual differences in pain responding lie on a continuum spanning extremes of confrontation and avoidance (3). Individuals who confront their pain are considered more likely to resume physical and social activities adaptively, whereas those who respond to pain with significant anxiety and avoidance (e.g. catastrophic thinking and avoidance behaviour) are considered more likely to enter a self-perpetuating vicious cycle that maintains and exacerbates pain perception, leading to chronic pain and related disability.

In the stress literature, Snyder (4) has argued that research findings indicate that coping with stress generally takes one of two routes, avoidance or approach. He noted that the weight of evidence demonstrates that the avoidance coping pathway is not adaptive, with a few important exceptions. Hayes and colleagues (5) have argued that higher levels of 'experiential avoidance', a type of avoidant coping, are associated with higher levels of general psychopathology, depression, anxiety, specific fears, trauma, and a lower quality of life.

The anxiety literature has demonstrated that short exposure to anxiety-provoking stimuli results in increased subsequent anxiety responses to the stimuli, whereas prolonged exposure to anxiety-provoking stimuli results in decreased subsequent anxiety responses (6). It is short exposure, resulting from attempts to avoid or escape from anxiety-eliciting situations, that underlies the maintenance of fears and phobias. In contrast, exposure-based approaches such as systematic desensitization, flooding and implosion, have been used with great success to treat a wide range of anxiety

disorders (7). Moses and Barlow (8) have recently proposed a new unified treatment approach for emotional disorders (defined as the anxiety and unipolar mood disorders), which includes preventing emotional avoidance (including both behavioural avoidance and cognitive avoidance), and facilitating emotional exposure.

A number of studies have investigated hypotheses with respect to headache triggers derived from the anxiety literature. Philips and Jahanshahi (9) proposed that exposure to a salient pain-provoking stimulus would lead to increasing tolerance through a process of adaptation, while avoidance of the stimulus would increase the potency of such stimuli to provoke pain. To test these predictions, chronic headache sufferers were exposed to noise. The results showed that exposure under optimal conditions (relaxation) was effective in reducing pain behaviours, whereas avoidance of exposure to potent stimuli led to increasing intolerance.

Martin (10) exposed headache and non-headache participants to the headache trigger of 'visual disturbance' (flicker, glare and eyestrain) for one of five durations ('none', 'very short', 'short', 'long' and 'very long'). Reports of head pain significantly differed as a function of length of exposure to the trigger. Nociceptive response was greater for the 'short' exposure condition than the 'none' and 'very short' exposure conditions; but the nociceptive response in the 'very long' condition was less than in the 'short' condition, and more approximated the nociceptive response in the 'none' and 'very short' conditions. In summary, the results were in line with the anxiety literature in that short exposure increased nociceptive response whereas very long exposure decreased nociceptive response. The study was repeated for the headache triggers of noise (11) and stress (12) with similar results.

Martin (13) completed a study in which six headache sufferers (four migraine and two tension-type headache) repeatedly attended the laboratory for sessions involving exposure to visual disturbance, paired with relaxation in some sessions. All participants had suffered from regular headaches for > 10 years, and all participants reported that visual disturbance was a trigger for their headaches. The results demonstrated that repeated, prolonged exposure to this trigger led to desensitization. With six sessions of exposure, ratings of visual disturbance, negative affect and headache intensity in response to the trigger decreased from baseline by 44, 54 and 63%, respectively. Participants kept diaries of their headaches during the period of the study in which they recorded hourly ratings of head pain throughout the waking day, and analysis of the diary data revealed that average pain ratings dropped by 19%. This is consistent with substantial desensitization to one headache trigger while other triggers retained their capacity to precipitate headaches.

So what are the implications of the above arguments and empirical findings? With respect to the aetiology of primary headache disorders, they suggest that one aetiological pathway is via trying to avoid or escape from triggers leading to short exposure and subsequent increased sensitivity or decreased tolerance of the triggers. Is this avoidance (environmental) model inconsistent with the dominant view, which emphasizes the role of genetics? Of course, the answer is that it is not. Some individuals are no doubt more susceptible to developing headaches via this pathway than others. The learning literature is replete with examples of interactions between genetic and environmental factors. For example, the anxiety literature talks about 'fears born and bred', i.e. innate vs. learnt fears (see Special Issue, *Behaviour Research and Therapy*, 40(2), 2002). Conditioning theories place emphasis on the environment (fears are learnt) but accept the role of heredity in determining which stimuli are most likely to become fear-provoking and who is most likely to learn to fear. Non-associative theories suggest that we are 'born to fear', but still acknowledge the role of the environment in accounting for the fact that many individuals subsequently learn not to fear, that is, habituation occurs through 'safe exposure'.

With respect to management of headaches, does this literature suggest that we should radically change direction and encourage exposure to triggers rather than avoidance? Of course, such an extreme position is not defensible, as indiscriminate exposure to triggers would simply lead to a lot of headaches, and who would advocate exposure to a headache trigger such as car exhaust fumes? However, the current approach of counselling avoidance of all triggers does not make much sense either. The most common trigger of migraine and tension-type headache, in both women and men, in both children and adults, is stress, and no expert on stress would suggest that the best approach to stress management is to try to avoid all stressors. In fact, exposure to less threatening stressors, so that individuals can practise coping skills in responses to stress, is a key component of many established stress-management techniques such as stress inoculation training. Anxiety is a common trigger of headaches, and decades of research demonstrate that avoidance of anxiety-eliciting situations leads to increased anxiety in response to those situations. Having said that, it still may make sense for headache sufferers to avoid specific situations that lead to extreme stress/anxiety.

Consideration of the literature on coping with stress provides some insight into managing headache triggers. Stress researchers have identified a range of stress-coping strategies that can be organized hierarchically. For example, Tobin et al. (14) identified eight primary factors (problem solving, cognitive restructuring,

emotional expression, social support, problem avoidance, wishful thinking, self-criticism, and social withdrawal), four secondary factors (combinations of problem-focused vs. emotion-focused, and engagement vs. disengagement), and two tertiary factors (approach and avoidance coping). The stress literature demonstrates that no single coping strategy can be selected as the best way of coping with stress for all situations and across time, but reviewers of this literature have been able to draw some conclusions, such as approach strategies generally are more adaptive than avoidance strategies, and more specific conclusions, such as avoidance can work better in acute situations but the opposite approach of attention to threat is more adaptive in the longer term (15).

It is suggested here that the philosophy of 'avoidance of triggers' be replaced by the philosophy of 'coping with triggers' because the latter philosophy is broader. One way of coping is avoidance and sometimes this will be the strategy of choice, but there are other coping strategies involving exposure, confronting, approach and engagement that should also be considered, and will often be more appropriate. Simply advising avoidance is tantamount to encouraging headache sufferers to 'cocoon' themselves away from anything that can cause a headache, which includes everything that is aversive, and sometimes more.

What are the advantages of planning controlled exposure to headache triggers? First, headache researchers are not certain about what can precipitate a headache, as most of the studies on triggers rely on retrospective self-reports, which are notoriously unreliable—only a few triggers, such as stress, visual disturbance, noise and hunger, have been experimentally validated. Headache sufferers often report their triggers incorrectly. This can happen for a number of reasons. For example, their blood sugar levels drop, which encourages them to eat certain foods which they then identify as the headache trigger, when it was the low blood sugar levels that really precipitated the headache. Sometimes, headache sufferers conclude that a factor precipitated a headache and then proceed to avoid that factor when if they continued to expose themselves to that factor they would realise that it does not cause headaches. Hence, one advantage of encouraging exposure is that it may reveal that the 'trigger' is not a trigger. Given that the evidence for food as a significant headache trigger is equivocal (16), this approach seems particularly worthwhile with 'food triggers'.

The main advantage of controlled exposure is to try to attenuate the response to the trigger, whether this is best described as developing coping resources, building tolerance, desensitization, habituation, adaptation, or extinction. There are well-established techniques for doing this with stress and emotional triggers.

The studies by Martin and colleagues suggest that exposure may be helpful for sensory triggers also. Triggers such as hunger and lack of sleep are interesting ones from the perspective of how to manage. It makes no sense to encourage missing meals and sleep, but if individuals are so determined to avoid headaches that they follow obsessional eating and sleeping routines then their bodies do not have a chance to learn to adapt to variations in eating and sleeping schedules.

Perhaps a combination of approaches is sometimes warranted, e.g. exposure to confirm the status of a trigger followed by further exposure to attempt to desensitize. If desensitization is not successful, avoidance may then be the last resort.

It could be argued that the above is speculative and lacks a strong evidence base. This is true, but so does the traditional approach of encouraging trigger avoidance. Clinicians could say that they have counselled avoidance and had patients report reduced headaches, but such advice will usually have been accompanied by other treatment strategies. Also, the arguments forwarded in this paper would suggest that the short-term effects of avoidance would probably be fewer headaches; the problem is the longer-term effects that may result from an insidious sensitization process.

Headache triggers are a neglected research domain. Research is needed on fundamental aspects of triggers, such as how do triggers interact, and do different triggers precipitate headaches via the same or different neurobiological mechanisms? The anxiety literature provides interesting concepts to explore with headaches. For example, can headache triggers be divided usefully into innate vs. learnt (conditioned) triggers? Anxiety sensitivity (fear of anxiety) has been shown to be a cognitive vulnerability factor for anxiety disorders, and fear of pain has been implicated in the development and maintenance of chronic pain behaviour. Would 'headache sensitivity' (fear of headache) be a cognitive vulnerability factor for headache disorders, perhaps mediated by trigger avoidance? Of course, randomized control trials are needed into how best to manage headache triggers. If this research unfolds, then perhaps in the fourth edition of our 'bible' (17), one out of 140 chapters will be devoted to the triggers of headaches rather than one out of 1169 pages as in the current edition!

Meanwhile, clinicians need to approach managing headache triggers with more flexibility than simply counselling avoidance of all triggers—think 'coping with triggers'.

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