



Behavioral management of headache triggers: Avoidance of triggers is an inadequate strategy

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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 review challenges that advice from a number of perspectives, including: that the advice is given in a theoretical vacuum; it is associated with practical problems; and it is not evidence-based. The review considers cognate literatures on stress, negative affect, and chronic pain that advocate approach/confront strategies over avoidance strategies. It is suggested that advice to avoid triggers could result in maintenance of the capacity of the trigger to precipitate headaches or even a sensitization process whereby tolerance diminishes. As anxiety researchers have investigated extensively the issue of how stimuli acquire and lose their capacity to elicit fear, this literature is explored to draw inferences for headache triggers. The review concludes with suggestions concerning etiology of chronic headache and associated management implications, and directions for future research. It argues that the philosophy of 'avoidance of triggers' should be replaced with 'coping with triggers,' as the latter includes both avoidance and approach/confront strategies involving exposure to triggers.

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It has been estimated that globally, percentages of the adult populations with an active headache disorder are 46% for headache in general, 11% for migraine, 42% for tension-type headache, and 3% for chronic daily headache (Stovner et al., 2007). These authors calculated that on the ranking of causes of disability of the World Health Organization (WHO), this would bring headache disorders into the 10 most common disabling conditions for the two genders, and into the 5 most common disabling conditions for women. Solomon, Skobierander, and Gragg (1993) concluded that patients with chronic headache have a level of functioning worse than patients with such chronic conditions as arthritis, diabetes, and back problems. The only chronic conditions that had similar levels of functional impairment to chronic headache were myocardial infarction and congestive heart failure, and the only disorder with worse levels of patient well being and functioning was symptomatic HIV infection. In attempts to rank severity of different diseases, migraine has been ranked among those causing the greatest degree of handicap, together with conditions such as quadriplegia, dementia and active psychosis (Dahlof & Solomon, 2006).

Headache disorders account for approximately 9% of lost labor productivity in the USA annually (Stewart, Ricci, Chee, Morganstein, & Lipton, 2003). The direct and indirect costs of migraine are high, with an estimated annual societal cost in the USA of US\$16.6 billion (Berg & Ramadan, 2006), and €27 billion in Europe (Olesen, Lekander, Andalin-Sobocki, & Jonsson, 2007). Equivalent figures are not available for tension-type headache but it is generally acknowledged that because this type of headache is much more common than migraine, it has a higher socioeconomic impact (Olesen & Welch, 2006).

Migraine attacks and tension-type headaches usually occur in response to identifiable triggers (Kelman, 2007). Headache triggers have been defined as “factors that, alone or in combination, induce headache attacks in susceptible individuals” (Zagami & Bahra, p. 400, 2006). Triggers (also called precipitating factors) usually precede the attack by less than 48 h. The most commonly reported triggers are: stress and negative emotions; hormonal factors for females; flicker, glare and eyestrain; noise; odours; hunger and consumption of certain foods and alcohol; weather; fatigue; and lack of sleep (discussed in detail below).

There is a long tradition in the clinical management of individuals who suffer from recurrent headaches to emphasize the importance of identifying the triggers and then avoiding them. For example, in writing about treatment of migraine and tension-type headache, Schulman and Silberstein (1992) state: “Identifying headache triggers is an extremely important part of headache therapy..... Wherever possible, practitioners should help susceptible patients learn to avoid triggers” (p. 16). Skaer (1996) advises that “migraine prevention is best achieved by avoidance of known migraine triggers” (p. 229). One of the ‘seven elements of good headache management’ listed by the WHO is “identification of predisposing and/or trigger factors and their avoidance through appropriate lifestyle change” (WHO, 2006, p. 77). Given that more people look for health information online in a typical day than consult a doctor (Fox & Rainie, 2002), it is also relevant to note that this advice appears on thousands of web sites ranging from the American Headache Society, through health web sites (e.g., Yahoo Health, BC Health Guide, Sutter Health), to Wikipedia. Trigger avoidance is sometimes referred to as ‘headache hygiene’.

The rationale behind this advice is clear, and if all headache triggers could be successfully avoided, then logically no headaches should occur. This strategy can be criticized, however, from conceptual, practical and empirical perspectives. Also, consideration of cognate research literatures on avoidance versus approach/confront as coping strategies for stress, negative affect (particularly anxiety), and chronic pain, casts further doubt on the wisdom of counseling avoidance of all triggers. It is crucial to consider how headache triggers have acquired the capacity to precipitate headaches and, conversely, how such capacity can be attenuated, as this is fundamental to understanding the impact of advocating avoidance. As the anxiety literature has considered extensively the questions of how stimuli and situations have acquired, maintained and lost the capacity to elicit anxiety, research in this domain may provide valuable clues for headache researchers.

1. Headaches and anxiety

Headache is defined as “pain located above the orbitomeatal line” (Headache Classification Subcommittee of the International Headache Society, p. 150, 2004). The International Association for the Study of Pain defines pain as “unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience” (Merskey, 1986, p. 1). The affective component of pain incorporates many different emotions with anxiety and depression, and recently anger, receiving the most attention in chronic pain patients. Anxiety is an aversive state of worry, fear, uneasiness, or apprehension “resulting from feelings of being unable to predict, control or obtain desired outcomes” (Barlow, 2000, 2002).

Headaches and anxiety are triggered by factors that are more or less easy to identify. Both can serve an adaptive function by signalling that self-protective action is required, but when occurring in the context of chronic disorders, this adaptive significance may be lost.

The precise nature of the relationship between headaches and anxiety is not of central relevance to this review, and hence will only be discussed briefly here. One way in which headaches and anxiety are functionally related is that anxiety is a common trigger of headaches. Most researchers investigating headache triggers do not attempt to separate stress, tension and negative emotions such as anxiety, as triggers, and clearly these factors are highly correlated (Nash & Thebauge, 2006). Nevertheless, when anxiety has been directly investigated as a trigger of headaches, it has emerged as one of the most common (Martin, Milech, & Nathan, 1993).

A second way in which headaches and anxiety are causally related is that anxiety is a common reaction to the experience of having a headache (Penzien, Holroyd, Holm, & Hursey, 1985; Philips, 1989). Individuals can react to headaches by worrying about a range of factors including the cause of the headache, how to manage it, and the impact it will have on them. Of course, this anxiety reaction often creates negative feedback loops whereby it increases the perception of pain, and exacerbates anxiety as a trigger factor (Martin, 1993).

A third way in which headaches and anxiety are related is that headache sufferers tend to be anxious individuals. Individuals who suffer from either migraine or tension-type headache score higher than headache-free controls on anxiety inventories (e.g., Andrasik & Holroyd, 1980; Hatch et al., 1991). Parenthetically, headache sufferers

also score higher than headache-free controls on scales measuring other types of negative affect such as depression and anger (Hatch et al., 1991; Martin, Nathan, Milech, & van Keppel, 1988).

A fourth way in which headaches and anxiety are related pertains to co-morbidity. Recent reviews have concluded that migraine is associated with anxiety disorders (particularly panic and phobia) (Radat & Swendsen, 2004; Sheftell & Atlas, 2002). Breslau (1998) reported that migraineurs were 4 to 5 times more likely to suffer from generalized anxiety disorder, and 3 to 10 times more likely to suffer from panic disorder. Fewer studies have investigated tension-type headache and psychiatric co-morbidity, but when studies have included individuals with migraine and individuals with tension-type headache, no differences in patterns of psychiatric co-morbidity have emerged between these two headache types (e.g., Marazziti et al., 1995).

Psychiatric co-morbidity is not restricted to anxiety disorders, and there also is a strong association between migraine and major depression (Radat & Swendsen, 2004; Scheftell & Atlas, 2002). Several studies have reported that, while the onset of anxiety typically precedes the onset of migraine, the onset of depression more often follows the onset of migraine (Breslau & Davis, 1993; Merikangas, Merikangas, & Angst, 1993).

Finally, increased anxiety in headache sufferers is associated with greater disability, poorer quality of life, and increased cost of care (e.g., Harpole et al., 2005; Lanteri-Minet, Radat, Chautard, & Lucas, 2005). Smith and Nicholson (2006) reported that less anxious/worried feelings after six months was a stronger predictor of lower headache impact than were changes in headache frequency or changes in medication.

2. Headache triggers

2.1. How many triggers can be identified by individual headache sufferers?

Epidemiological studies indicate that approximately 85% of headache sufferers report at least one significant identifiable trigger (Paulin, Waal-Manning, Simpson, & Knight, 1985; Van den Bergh, Amery, & Waelkens, 1987). Van den Bergh et al. (1987) indicated that migraineurs reported a median of 3 triggers with a range from 1 to 12. Blau and Thavapalan (1988) reported an average of 4 triggers volunteered by migraineurs, a number that increased to 5.5 with direct questioning. A recent large, clinical study by Kelman (2007) found that 76% of migraineurs responded affirmatively when asked whether they had triggers for migraine attacks, but this figure rose to 95% when individuals were asked to respond to a specific list of triggers. This study found the mean number of triggers reported per patient to be 6.7 with almost two-thirds of patients reporting 4 to 9 triggers.

2.2. What are the triggers of headaches?

Blau and Thavapalan (1988) note that an often expressed view is that “everything can produce a migraine” (p. 483). The precipitants listed in this study, by two other prominent clinicians in earlier review articles (Diamond, 1979; Saper, 1978), and more recent papers cited subsequently in this review, include stress and emotional factors, post-stress relaxation, exercise, fatigue, and too much or too little sleep. Foods often considered to be triggers of migraine include ripened cheeses; herring; chocolate; vinegar; anything fermented, pickled, or marinated; sour cream and yogurt; nuts and peanut butter; hot fresh breads, raised coffee cakes, and doughnuts; pods of broad beans; any foods containing large amounts of monosodium glutamate; onions; canned figs; citrus foods; bananas; pizza; excessive tea, coffee, and cola beverages; avocado; fermented sausages; and chicken livers. All alcoholic beverages are potential precipitants of headaches, particularly red wine and beer (Peatfield, 1995). It has been suggested that withdrawal of reactive foods, particularly caffeine, may worsen

headaches in the short-term. Hunger is considered to be an important trigger (skipped meals, delayed meals, fasting) (Blau & Cumings, 1966), and it also has been suggested that dehydration can precipitate headaches (e.g., Blau, Kell, & Sperling, 2004).

Askmark, Lundberg, and Olsson (1989) reported a survey of drug-related headaches in which indomethacin (a nonsteroidal analgesic and anti-inflammatory agent used in the treatment of disorders such as rheumatoid arthritis and osteoarthritis) and nifedipine (a calcium antagonist used in the treatment and prevention of angina pectoris) were the drugs most frequently implicated as triggers of headaches. It has been suggested that headaches can be triggered by smoking cigarettes (Blau & Thavapalan, 1988).

Hormonal factors can be important for female sufferers including menstruation, menopause, pregnancy, use of oral contraceptives, and hormone replacement therapy (HRT) (Silberstein, 1999). Menstruation seems particularly significant – headaches may become more frequent before, during, or after periods. The relationship between headaches and hormonal changes is complex and not entirely understood. For example, migraines tend to get worse in early pregnancy and improve as pregnancy progresses. HRT can make migraine worse but occasionally is associated with improvement. Migraine in women often improves after menopause but this is not always the case.

Sensory factors that can act as headache triggers include visual stimuli such as glare, flicker, and eyestrain; noise; and odours/smells (e.g., paint, exhaust fumes, cleaning solutions, perfume). Diamond (1979) drew attention to certain workers who are likely to suffer chronic headaches due to exposure to potentially toxic substances (e.g., auto mechanics who inhale carbon monoxide in a poorly ventilated garage). Seasonal and meteorological precipitants include cold, heat, high humidity, and sudden changes in weather. Other triggers include allergens such as pollen, neck pain, head trauma, coughing and sneezing, sexual intercourse, crying and laughing.

Several studies have quantified the percentage of headache sufferers who report different triggers of their attacks. The results from five recent studies are presented in Table 1. The studies are: (i) Kelman

Table 1
Percentages of headache patients reporting triggers in five recent studies.

Headache triggers	Migraine					Tension-type Headache	
	A	B	C	D	E	D	E
Stress/tension	80	71	76	84	80	82	97
Menstruation	65	31	39	57	59	38	32
Visual disturbance	38	32	75 ^a	50	27	18	3
Noise		42	75 ^a	53			29
Odours/smells	44	26	75 ^a	61	29	24	0
Hunger/not eating	57	42	48	82	73	76	39
Specific foods/drinks	27	33	46	58	34	35	7
Alcohol	38	28		42	4	29	7
Weather	53	17	71	48	35	36	
Heat	30						
Lack of sleep	50	48	49	74	54	71	26
Sleeping late/excess	32		27		25		13
Exercise	22		20	42	20	35	16
Fatigue/tiredness		22	35	79		65	
Straining				42		24	
Sexual activity	5	1	3		7		10
Head/neck movements			2		9		48
Neck pain	38						
Head trauma			20				
Coughing/sneezing				7		24	
Smoking		16	1				
Smoke	36			61		29	
Travel/trips/driving		9	4	29		12	

Note. A – Kelman (2007); B – Deniz et al. (2004); C – Ierusalimsky & Moreira Filho (2002); D – Spierings et al. (2001); E – Karli et al. (2005).

^a Ierusalimsky & Moreira Filho (2002) presented a combined figure for ‘sensorial stimuli’ of 75%.

(2007) – clinical sample from the USA of 1207 individuals diagnosed as migraine with or without aura, and chronic migraine; (ii) Deniz, Aygul, Kocak, Orhan, and Kaya (2004) – clinical sample from Turkey of 185 individuals diagnosed as migraine with or without aura; (iii) Ierusalimschy and Moreira Filho (2002) – clinical sample from Portugal of 100 individuals diagnosed as migraine without aura; (iv) Spierings, Ranke, and Honkoop (2001) – clinical sample from the Netherlands of 38 individuals diagnosed as migraine and 17 individuals diagnosed as tension-type headache; and (v) Karli, Zarifoglu, Calisir, and Akgoz (2005) – clinical sample from Turkey of 31 tension-type headache, 23 migraine with aura, 33 migraine without aura, and 9 with typical aura with non-migraine headache (the latter group was omitted from the Table and the two migraine groups were combined). A similar synoptic table for eight earlier studies on migraine triggers is presented in Zagami and Bahra (2006).

Inspection of Table 1 reveals that stress/tension is the most widely endorsed headache trigger, and this same conclusion has been drawn in many other studies (e.g., Blau, 1990; Martin et al., 1993). Stress is the most important trigger factor for both males and females (Robbins, 1994), and for children as well as adults (Osterhaus & Passchier, 1992). Menstruation is an important trigger factor for female headache sufferers. The role of the sensory factors, visual disturbance, noise and odours/smells, is clear in the Table and reinforced by other studies, such as Harle, Shepherd, and Evans (2006) on visual disturbance, and Martin et al. (1993) and Blau (1990) on noise. Other factors that stand out as being common headache triggers are hunger and lack of sleep.

2.3. Groupings of triggers and how triggers relate to diagnosis

Martin et al. (1993) used discriminant function analysis to explore whether headache sufferers could be meaningfully grouped according to the reported triggers of their headaches, and found this not to be the case. They then used factor analysis to investigate whether triggers aggregated into meaningful patterns and observed that they did. Five categories of headache triggers emerged: Negative Affect (stress, anxiety, depression, and anger); Visual Disturbance (flicker, glare, and eyestrain); Somatic Disturbance (sneezing, coughing, and pollen); Environmental Stress (humidity, high temperature, and opposite to relaxation); and Consummatory Stimuli (alcohol, food, and hunger).

Several studies have investigated whether migraine and tension-type headache 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). However, when Spierings et al. (2001) compared these two diagnostic groups on 18 precipitating factors, they observed that the triggers of weather, odours/smells, smoke and light, were more commonly reported by individuals with migraine than tension-type headache (see Table 1). Karli et al. (2005) also compared individuals suffering from migraine and tension-type headache on 18 trigger factors and found that hunger and odours/smells were more common triggers in the migraine group, and head and neck movements were more common triggers in the tension-type headache group (see Table 1). In their factor analytic study, Martin et al. (1993) found tension headache and migraine sufferers not to differ on the factors of Negative Affect or Environmental Stress, but migraineurs scored higher on Visual Disturbance, Somatic Stress, and Consummatory Stimuli.

2.4. Interactions between triggers

Triggers may not operate independently. Occurrence of one trigger may increase the likelihood of exposure to one or more other triggers. For example, monthly hormonal changes in women may increase the probability of the consumption of certain foods (O'Banion, 1981). Alcohol consumption and smoking cigarettes commonly occur together.

The simultaneous occurrence of more than one trigger may result in an aggregative effect, and indeed some candidate triggers may not

be capable of eliciting headaches without the presence of an additional trigger, at least for some individuals. A number of investigators have suggested that alcohol and food are more likely to trigger migraines at some times than at others (e.g., Dalton, 1975; Saper, 1978). Blau and Thavapalan (1988), for example, note that some women know that they can drink wine without inducing an attack except during the premenstrual week, and O'Banion (1981) argued that diet may influence the severity of problems produced by monthly menstrual cycles. Nicolodi and Sicuteri (1999) found that low doses of alcohol were much more likely to lead to migraines when consumed on stressful occasions. In his headache trigger model, Scopp (1992) contends that “the additive effect of several potential triggers occurring in close temporal proximity results in an increased probability of a cascade-like precipitation of migraine” (p. 32). Others have suggested that simultaneous exposure to more than one trigger factor can result in a multiplicative effect rather than an additive one (Leviton, 1984). Sauber (1980), for example, described the potentiating effect alcohol had on his headache vulnerability following consumption of monosodium glutamate (“Chinese restaurant” syndrome).

Only two published studies have experimentally investigated interactions between triggers (Martin & Seneviratne, 1997; Martin, Todd, & Reece, 2005), and these studies will be discussed in more detail in the next section. Suffice to say here that Martin and Seneviratne (1997) found no evidence to support an interactive influence of stress and hunger, while Martin et al. (2005) found no evidence to support an interactive influence of stress and noise on headache symptomatology.

The large number of identified headache triggers raises the question of whether triggers precipitate headaches by the same or different biological pathways? Martin and colleagues have addressed this question in a series of studies, and have reported different peripheral physiological responses to a stressor and visual disturbance (Martin & Teoh, 1999), a stressor and noise (Martin et al., 2005), and a stressor and hunger (Martin & Seneviratne, 1997). The authors conceded, however, that the results could not conclusively establish whether these triggers impacted on headaches via a common or different biological pathways, as the different physiological responses recorded did not map onto the most plausible alternative peripheral mechanisms considered to be implicated in the genesis of headaches (e.g., vascular versus muscular).

In contrast, Burstein and Jakubowski (2005) have hypothesized that different migraine triggers, such as stress, perfume and awakening, activate a wide variety of brain areas, but that there is a common descending pathway accounting for the activation of meningeal nociceptors. They argue that the perception of migraine headache arises from the nociceptive signals originating in the meninges.

2.5. What is the research evidence in support of triggers?

Most of the research on triggers is based on retrospective self-report, and hence the validity of the data is challenged by the possibility of selective memory and the patient's need for causal explanations (Wober et al., 2007). Fortunately, some studies have taken a prospective approach, and some have experimentally investigated whether candidate triggers can truly precipitate headaches by manipulating these factors and comparing pain responses with those observed under control conditions. In prospective studies, several researchers have instructed headache sufferers to self-monitor headaches and stress, and have reported positive relationships between the two (e.g., Kohler & Haimerl, 1990; Sorbi & Tellegen, 1988). Other investigators have requested headache sufferers to self-monitor headaches and mood, and have reported that headaches occur in response to a range of negative moods, such as anxiety, depression, hostility, and tiredness (e.g., Arena, Blanchard, & Andrasik, 1984; Martin et al., 1988). Some evidence was obtained in two early prospective studies (Dalton, 1975; Medina & Diamond, 1978) that headaches can occur in association with eating certain foods (particularly cheese and chocolate), consumption of alcohol, or hunger. A large prospective analysis was reported recently by

Wober et al. (2007). They instructed 327 migraineurs to self-monitor their headaches and precipitating factors over a period of three months. Menstruation emerged as the most important trigger factor. Other significant trigger factors included stress/tension, tiredness, noise, and odours/smells. Johannes et al. (1995) reported a significantly increased risk (1.66 times as great) of having a migraine without aura during the first three days of menstruation compared with other days.

Manipulation studies have lent further support to the hypothesized role of triggers in precipitating headaches. Haynes and colleagues exposed headache sufferers to prolonged stress and reported that this served to elicit headaches in 69% (Gannon, Haynes, Cuevas, & Chavez, 1987) and 83% (Haynes, Gannon, Bank, Shelton, & Goodwin, 1990) of their samples. Martin et al. (1988) used a mood induction procedure to modify mood state and reported a significant decrease in headache intensity associated with induced positive mood and a trend toward an increase in headache activity associated with inducing depressed mood. Martin and colleagues have demonstrated across several studies that experimentally-induced Negative Affect can elicit reports of head pain (Martin & Seneviratne, 1997; Martin & Teoh, 1999; Martin et al., 2005).

Martin and colleagues have also experimentally validated a number of other commonly cited headache triggers, using the manipulation approach. Martin and Teoh (1999) induced 'visual disturbance' (flicker, glare and eye strain) by having participants try to read small font words in a dark room with a bright stroboscopic light flashing in the background. They reported that such induced visual disturbance was associated with elevated head pain compared to a control condition. In another study, 50 dB of white noise was demonstrated to induce head pain (Martin et al., 2005), as was hunger induced by 19 h of food deprivation (Martin & Seneviratne, 1997). Blau and Cumings (1966) also reported evidence that hunger was associated with headaches.

In contrast to these positive findings supporting the headache eliciting capability of some triggers identified by self-report, negative findings have been reported for supposed dietary triggers. Moffett, Swash, and Scott (1974) completed two double-blind studies investigating chocolate as a precipitant of migraine, and the results suggested that chocolate on its own is rarely a trigger. Medina and Diamond (1978) assigned participants to three different dietary regimes and found no difference in terms of headaches reported. Koehler and Glaros (1988) did report an association between consuming aspartame (dietary sweetener) and headache, but Schiffman et al. (1987) found no such association even using daily doses of aspartame equivalent to the amount found in 4 l of diet soda. Hannington (1967) argued that a person's retrospective report of the association between dietary factors and headache experience was likely to be spurious, an artefact of what they read, or of illusory correlations, and she reserved the term 'dietary migraine' for individuals who had actually altered their diet to avoid headaches. She estimated that only 5% of migraineurs would meet this definition, and the results of manipulation studies provide no reason to consider this an overly conservative estimate.

3. The case against counseling avoidance of all headache triggers

3.1. Conceptual, practical and empirical criticisms

Despite frequency with which it is given, clinical advice to avoid headache triggers has emerged from a theoretical vacuum, rather than resulting from an explicitly formulated and empirically verified theory concerning how a trigger precipitates a headache. Hence, the advice takes no account of the potentially negative impact of changing the exposure pattern to triggers such as the undesirable possibility that reduced exposure, resulting from attempts to avoid, will decrease tolerance for these triggers, thereby increasing potential to elicit headaches. Researchers write about triggers as though the capacity to precipitate a headache is an inherent and immutable property of

the trigger itself, with individual differences in sensitivity to triggers considered to be genetically-determined (e.g., Bussone, 2004; Nedeltchev et al., 2004). This conceptualization of headache triggers is problematic in a number of ways. For example, studies have shown that monozygotic twins are concordant for migraine in only 20% to 50% of cases (Russell & Olesen, 1995), so that at least 50% of those carrying a genetic predisposition to migraine never experience attacks. Also, apparent carriers of this disposition express migraine only during part of their lifetime because the prevalence of migraine increases up to age 40 and decreases in old age (Lipton, Stewart, Diamond, Diamond & Reed, 2001). Bille (1997) showed in a longitudinal cohort study that children with migraine often became migraine-free for years or decades, but many later had a recurrence of attacks. If the capacity of a trigger to precipitate a headache is invariant, then headaches 'coming and going' across the life span can only be explained by synchronous changes in the presence or absence of triggers, and this seems unlikely to account fully for all observed temporal variations in the occurrence of headaches.

From a practical perspective, there are problems associated with the advice to avoid all triggers. It is unlikely that it could prove possible to completely avoid all potential headache triggers; and attempting to do so could result in a very restricted lifestyle (Kelman, 2007). Marcus (2003) pointed out that the effort to avoid every potential headache trigger may itself be stressful. Furthermore, advice to avoid triggers 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. This could exacerbate a headache disorder, as Marlowe (1998) has reported that the correlation between frequency of headaches and frequency of stressful events is moderated by self-efficacy; the more a person felt capable of handling stressors effectively, the lower was the observed correlation between headaches and stress.

Empirical evidence supporting therapeutic efficacy of avoiding headache triggers is very limited, and the few studies suggesting clinical benefits from this approach suffer from serious methodological limitations. Grant (1979) reported on 60 migraineurs who completed elimination diets and were advised to avoid stress, hunger and cigarette smoke, and claimed that there was a dramatic fall in the number of headaches per month with 85% of patients becoming headache-free. Blau and Thavapalan (1988) encouraged migraineurs to avoid all precipitating factors and reported a reduction of 50% in attack frequency in 19 out of 23 patients. A few case studies also have reported successful dietary treatment of headache. O'Banion (1981) reported five case studies of individuals experiencing migraine, tension headache, or combined headaches treated by controlling dietary factors. Headaches were eliminated in four of five cases. Scopp (1991) treated two individuals, both suffering from migraine and tension headache, by eliminating all food sources containing monosodium glutamate. Marked reductions in the frequency of both types of headaches were reported for both cases. Radnitz, Blanchard, and Bylina (1990) treated two migraineurs who had failed to respond to stress management training via a dietary approach. After elimination of trigger foods, both individuals showed a 50% decrease in headache activity. Dalton (1973) reported one migraineur who responded to eliminating pickles from his diet, and another who responded to eliminating chocolates from her diet.

Methodological criticisms of these studies include the following. Neither of the two group studies included control conditions. Furthermore, both these studies were compromised by measurement problems. Grant (1979) 'estimated' headaches over a three-month period before and after the intervention, without carefully measuring headache incidence across this period. Blau and Thavapalan (1988) compared a retrospective estimate of attack frequency in the three months before consultation with 'noting attacks' during the two months after consultation, meaning that pre versus post intervention differences could potentially be attributable to the differing measurement

approaches employed on each occasion. Patients in the Grant study were consuming ergotamine at a rate that far exceeded the criterion for 'ergotamine-overuse headache' (average of 115 tablets per month) prior to the intervention, but not after the intervention, so improvement may have occurred as a consequence of no longer experiencing ergotamine-induced headaches. Blau and Thavapalan advised their patients "how to abort attacks by quickly taking an anti-nauseant and analgesic tablets" (p. 481), so that the effects of this advice were confounded with the effects of the advice to avoid precipitants. None of the case studies on avoidance of triggers referred to above included any within-subject control conditions. None of the studies assessed treatment adherence, so it is not clear the degree to which participants followed the advice. None of the studies included long-term follow-up, which is a problem, as this review will suggest that the short-term response to advice to avoid triggers may well be a decrease in headaches, but the long-term response may be an increase.

3.2. Avoidance versus approach/confront as coping strategies for stress, negative affect and chronic pain

Avoidance is clearly an appropriate strategy when faced with some 'threatening' situations but empirical evidence shows it to be maladaptive in many situations. Snyder (2001) 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. In a review written over 30 years ago, Meichenbaum (1977, p. 149) argued that "actual exposure during training to less threatening stressful events has a beneficial effect." This principle was incorporated into stress inoculation training (Meichenbaum, 1985), which has been applied successfully to a diverse array of clinical disorders including phobias and other anxiety-related disorders, anger and pain, in addition to stress.

Stephen Hayes and colleagues have written extensively about the problems associated with 'experiential avoidance,' a form of avoidant coping. They define experiential avoidance as "the phenomenon that occurs when a person is unwilling to remain in contact with particular private experiences (e.g., bodily sensations, emotions, thoughts, memories, images, behavioral predispositions) and takes steps to alter the form or frequency of these experiences or the contexts that occasion them, even when these forms of avoidance cause behavioral harm" (Hayes et al., 2004, p. 553). They have argued that higher levels of experiential avoidance are associated with higher levels of general psychopathology, depression, anxiety, specific fears, trauma, and a lower quality of life. A variety of behavior therapy approaches begun exploring methods for changing experiential avoidance by, for example, encouraging clients to directly experience problematic emotions (Blackledge & Hayes, 2001). These include Dialectical Behavior Therapy (Linehan, 1993), Mindfulness-based Cognitive Therapy (Segal, Williams, & Teasdale, 2001), Acceptance and Commitment Therapy (Hayes, Strosahl, & Wilson, 1999), and Integrative Couples Therapy (Jacobson & Christensen, 1996). Initial results with these approaches have been positive.

In the anxiety literature, Mowrer's classic two-factor theory of anxiety proposed that fears are acquired through classical conditioning, but are maintained because they are protected from extinction by subsequent escape and avoidance of phobic objects, itself reinforced by contingent transient attenuation of anxiety (Mowrer, 1960). Over the years, the first factor in this theory has been criticized on the grounds that classical conditioning is only one explanation for how fear develops, but the role of avoidance in the maintenance of fears remains largely unchallenged (Tryon, 2005). There is now a range of treatments for anxiety disorders, including systematic desensitization, flooding and implosion, that have in common prolonged exposure to the anxiety-eliciting stimuli. These treatments have been shown to be highly effective (Barlow, 2002). Moses and Barlow (2006) 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 behavioral avoidance and cognitive avoidance), and facilitating emotional exposure. The expanding use of exposure-based therapies for psychopathology has led to the recent publication of the *Handbook of exposure therapies* (Richard & Lauterbach, 2006) which advocates the use of exposure to triggers in the treatment of disorders other than anxiety dysfunction (e.g., substance abuse).

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 (Asmundson, Norton, & Vlaeyen, 2004). Individuals who confront their pain are considered more likely to adaptively resume physical and social activities, whereas those who respond to pain with significant anxiety and avoidance (e.g., catastrophic thinking and avoidance behavior) are considered more likely to enter a self-perpetuating vicious cycle that maintains and exacerbates pain perception, leading to chronic pain and related disability. Lethem, Slade, Troup, and Bentley (1983), for example, hypothesized that whether the individual avoids or confronts pain may be instrumental in determining the chronicity of the pain or 'invalid status.' Fear of pain generates in some individuals a strategy of avoidance rather than confrontation, which in turn leads to both physical and psychological reinforcement of invalid status. Most of the chronic pain literature focuses on low back pain but Hursey and Jacks (1992) reported that recurrent headache sufferers showed much greater fear of severe pain than controls, findings that are consistent with fear-avoidance models of chronic pain.

There is a long history of using distraction (a form of avoidance) as a coping strategy for experimental and clinical pain, but the opposite strategy of focusing attention directly onto painful sensations has also been employed (Nouwen, Cloutier, Kappas, Warbrick, & Sheffield, 2006). An interesting literature is emerging about the neurological mechanisms that underpin attentionally mediated variations in subjective pain (cf Tracey, 2008; Villemure & Bushnell, 2002). Clinical studies comparing focused attention and distraction have yielded mixed results, with some finding no significant differences between the strategies (Cioffi & Holloway, 1993; Stevens & Terner, 1992), others finding distraction to be superior (Arntz, Dressen, & Merckelbach, 1991; Brewer & Karoly, 1989), and others finding focused attention to be superior (Ahles, Blanchard, & Leventhal, 1983; McCaul & Haugtvedt, 1982).

Parenthetically, it is interesting to note parallels between recurrent headaches and the related disorder of asthma (Aamodt, Stovner, Langhammer, Hagen, & Zwart, 2007). Both are chronic conditions characterized by periodic 'attacks' precipitated by triggers, with some triggers in common (e.g., stress and anxiety, exercise, and certain foods and medications) (American Academy of Allergy, Asthma and Immunology, 2008). Asthma is also associated with counseling to avoid triggers, for example, "Asthmatics should try and avoid exposure to substances that may trigger their asthma" (Prendergast, 1995, p. 40). However, a Cochrane review of the literature on allergen avoidance as a treatment for allergic asthma, concluded that there was no empirical support for the efficacy of this approach (Gotzsche, Johansen, Burr, & Hammarquist, 2003). After reviewing the literature on allergen avoidance, two of the leading researchers in this field asked, "are we going the wrong way?", and went on to conclude "Whether one should pursue the induction of tolerance by high dose allergen exposure 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" (Sporik & Platts-Mills, 2001, p. 61). Allergen immunotherapy consists of a vaccination program that involves administering gradually increasing amounts of an allergen to a patient over several months, and has been demonstrated in randomized control trials to be an effective treatment for allergic asthma (e.g., Abramson, Puy, & Weiner, 1995; Bousquet & Michel, 1994).

4. How do triggers acquire and lose the capacity to elicit anxiety?

Over the last century, starting with the work of Pavlov, Watson, and Freud, much attention has been given to the processes involved in acquisition of fears, persistence of fears, and extinction of fears. In the case of fears precipitated by exposure to triggers, this literature has been concerned with how stimuli acquire the capacity to elicit anxiety, maintain the capacity to elicit anxiety, and lose the capacity to elicit anxiety. This literature is extensive, but some of the major recurring ideas suggest plausible hypotheses concerning how headache triggers may acquire and lose their capacity for precipitating headaches.

In Mowrer's two-stage theory of fear and avoidance, he argued that fears initially are acquired by classical conditioning, but subsequently are maintained by the fear reduction that comes from escape and avoidance of the phobic stimulus (Mowrer, 1939, 1960). Stimuli and situations that previously did not elicit anxiety could come to evoke anxiety if paired with other stimuli or situations that elicit anxiety or pain (unconditioned stimuli). The strength of this conditioned fear is a function of the number of repetitions of the association between the unconditioned and conditioned stimuli, and the intensity of the fear or pain experienced in the presence of the unconditioned stimuli. According to classical conditioning theory, presentation of the conditioned stimulus without the unconditioned stimulus should lead to extinction of the conditioned response. As fears and phobias are often long-lasting, the second stage of the theory was the proposition that fear motivates avoidance of anxiety-evoking stimuli, thereby preventing extinction from occurring.

Theories such as Mowrer's focussed attention on the relationship between length of exposure to a feared stimulus and the maintenance of the stimulus' capacity to elicit anxiety. The anxiety literature demonstrated that short exposure to anxiety-provoking stimuli results in increased subsequent anxiety responses to the stimuli, whilst prolonged exposure to anxiety-provoking stimuli results in decreased subsequent anxiety responses (Eysenck, 1979). Such findings support the theory that short exposure to feared stimuli, resulting from phobics avoiding or escaping from these anxiety-provoking stimuli, underpins the maintenance of phobias. Consequently, treatment has become based on the reverse principle of prolonging exposure to fear triggers.

Rachman (1977) proposed that there were three pathways to fear: conditioning, vicarious exposures, and the transmission of information and instruction. Vicarious and informational transmission of fears can take place in the absence of direct contact with the fear stimulus. Vicarious acquisition of fears involves fear of situations developing as a consequence of observing an intense fear reaction to the situation expressed by others. Acquisition of fear by information and instruction proceeds through the information-giving that is an inherent part of child-rearing, carried on by parents and peers on a regular basis, particularly in the child's early years. Hence, fears are learnt via individuals being instructed as to what situations warrant a fear reaction.

In 2002, a Special Issue of *Behaviour Research and Therapy* was devoted to the issue of "fears born and bred." In this series, Poulton and Menzies (2002) argued the need for a fourth pathway to fear. They postulated that some fears are innate, suggesting that stimuli/situations associated with innate fears are ones that represent long-standing danger to the species; and that the fear and avoidance of such stimuli/situations is partly under genetic control (Menzies & Clarke, 1995). These authors argued that the role of the environment is to abate biologically-relevant fears, through extinction, rather than to precipitate their emergence. They proposed that innate fears are present at birth but will be maintained into adulthood only if individuals have not been exposed enough to the anxiety-eliciting stimulus for habituation to occur. On the basis of the four pathways to fear, Poulton and Menzies suggest that some fears may be learned, but others are innate fears that people have not learnt to overcome via "safe exposure."

The distinction between innate and learned fears ('fears born and bred') is an interesting one as it accords primacy in fear acquisition to

different factors. Nevertheless, the theories underlying the two types of fears have much in common. First, both types of fear are considered to involve an interaction between environmental and genetic factors. The pathways based on learning consider genetics important in various ways (e.g., which stimuli are most easily conditioned), and the innate fear pathway considers the environment critical with respect to whether the fear habituates. Second, both theories recognize the importance of individual differences. Conditioning theories accept individual differences in conditionability and non-associative theories acknowledge individual differences in rate or speed at which habituation takes place. Third, both theories view avoidance of, or lack of exposure to, the feared stimulus as critical in the maintenance of fear.

A number of very successful treatment approaches for anxiety have been developed, based on the conditioning theories. In 1958, Wolpe proposed systematic desensitization as a treatment for phobias (Wolpe, 1958). This technique included a number of different elements. It involved development of a hierarchy of imaginal, anxiety-provoking items ranging from items that evoked low levels of anxiety to items that evoked high levels of anxiety. The technique also involved relaxation training. Much subsequent research investigated which elements of this technique were contributing to therapeutic effectiveness and this led to the conclusion that exposure to the phobic stimulus was the only critical ingredient (Marks, 1975). Several variants of exposure-based treatments for anxiety disorders have been proposed. Taylor (2002) has categorized them on two dimensions: (i) real versus imagined stimuli; and (ii) gradual versus intense exposure. Hence, the four types of exposure therapy are: (i) systematic desensitization (imagined, gradual); (ii) implosion (imagined, intense); (iii) graded in vivo exposure (real, gradual); and (iv) flooding (real, intense). Research has shown that exposure-based therapies are effective across a wide range of anxiety disorders (Barlow, 2002), although it is still not clear why they are effective (Tryon, 2005).

5. Headache triggers in the context of the anxiety literature

A striking feature of the anxiety literature, in the context of understanding headache triggers, is that the theorizing always brings together environmental and genetic factors. 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.

Given that avoidance of anxiety-eliciting stimuli is accorded so much significance in the anxiety literature, as a maladaptive response that serves to maintain fears, it is reasonable to question whether headache sufferers try to avoid headache triggers. Available evidence suggests, not surprisingly, that typically they do. Philips and Hunter (1981) developed a Pain Behavior Checklist (PBC) and reported that such avoidance responses were endorsed frequently by both tension headache and migraine sufferers. Anciano (1986) administered a version of the PBC to headache sufferers and factor analyzed the results. Three factors emerged, the first of which was avoidance. Appelbaum, Radnitz, Blanchard, and Prins (1988) carried out a similar study and identified avoidance as the first factor.

Two studies have investigated how headache sufferers respond to the most common headache trigger, namely stress. Holm, Holroyd, Hursey and Penzien (1986) compared recurrent headache sufferers and matched headache-free controls on the Coping Strategy Inventory and reported that headache sufferers used avoidance (cognitive and behavioral avoidance of stressful situations) more than the controls. Ehde and Holm (1992) found that migraine sufferers reported using more problem avoidance in coping with stressful events than did

headache-free controls, and tension headache sufferers have a greater tendency than headache-free controls to cope with stressful events in ways emphasizing behavioral, cognitive, and emotional disengagement or avoidance.

An early study investigating the relationship between length of exposure to a headache trigger and the capacity of that trigger to elicit head pain, was carried out by Philips and Jahanshahi (1985). They 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 behaviors whereas avoidance of exposure to potent stimuli led to increasing intolerance.

Martin and colleagues have carried out a more recent series of studies to investigate whether length of exposure to a headache trigger affects the capacity of the trigger to elicit head pain. Based on the anxiety literature, Martin (2001) proposed two theories with respect to how individuals become sensitized to headache triggers. One position suggested that on each occasion a trigger is associated with a headache, the link between the two is strengthened or consolidated, as occurs through learning by practice or repetition (*repetition theory*). The alternative position was that perception by a headache sufferer of a link between a supposed trigger and a headache (whether the observation was accurate or inaccurate) results in escape/avoidance behavior whereby the sufferer seeks to minimize exposure to the trigger. Such avoidance sensitizes the individual to the trigger in the same way that avoidance of anxiety-inducing stimuli results in increased anxiety to future presentations of these stimuli (*avoidance theory*).

In Martin (2001), 110 participants were exposed to 'visual disturbance' for one of five durations ('none,' 'very short,' 'short,' 'long,' and 'very long'). Both theories predicted that short exposure would lead to increased sensitivity to the trigger. With respect to long exposure however, the repetition theory predicted further increases in sensitivity whereas the avoidance theory predicted decreased sensitivity. If headache response to the trigger were plotted against length of exposure, 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. In the sample, 48 participants suffered from 'regular' headaches (at least one per fortnight), and 62 participants did not suffer from regular headaches. There was no suggestion in the data of a differential response to exposure conditions as a function of headache status, and hence the data for all participants were analyzed together. 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 supported the avoidance theory rather than the repetition theory.

Martin, Reece, and Forsyth (2006) repeated the Martin (2001) study with 115 participants using noise as the trigger factor rather than visual disturbance. For this study, the alternative predictions from the two theories were tested using analyses of orthogonal polynomials (i.e., trend analyses). For participants who did not suffer from regular headaches, significant quadratic trends were found (inverted U-curves), supporting the avoidance theory. For participants who did suffer from regular headaches, the findings were less clear cut as there was a significant linear trend and a quadratic trend which tended towards significance ($p = .063$). The linear trend reflected increasing nociceptive response as a function of length of exposure and was consistent with the repetition theory. The tendency towards a quadratic trend reflected a pattern of responses whereby the

nociceptive response increased from 'none' to 'very short' to 'short', decreased to 'long', before increasing again to 'very long'. This trend was consistent with the avoidance theory.

Martin, Lae, and Reece (2007) repeated the Martin (2001) study with 127 participants using stress as the trigger. As for the original study, results did not differ between those who suffered from regular headaches and those who did not. Analyses of orthogonal polynomials revealed a significant cubic trend between length of exposure to the stressor and ratings of pain. This trend indicated that very short exposure to the stressor increased sensitivity whilst longer exposure decreased sensitivity, again supporting the avoidance theory; however, even longer exposure increased sensitivity, akin to the response to very long exposure to noise in the headache group in Martin et al. (2006).

It is interesting to note that in these three studies of headache triggers (Martin, 2001; Martin et al., 2006; Martin et al., 2007), the curvilinear relationship between length of exposure to a trigger and sensitivity to the trigger was not significantly different between headache sufferers and non headache controls for two triggers (visual disturbance and stress) and only differed marginally (significant versus a trend) for the third trigger (noise). This suggests that the curvilinear relationship is not a function of being a headache sufferer or having a predisposition to develop a headache disorder, but rather characterizes the relationship between length of exposure and sensitivity for everyone. It is consistent with the hypothesis that trying to avoid or escape from triggers resulting in short exposure, puts everyone at risk of developing a headache disorder via increased sensitivity to triggers.

As the Martin (2001) study indicated that long exposure to visual disturbance was associated with a decreased nociceptive response, Martin (2000) 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 more than 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 whilst other triggers retained their capacity to precipitate headaches.

Results of the five studies reviewed above are consistent with the proposition that the relationship between exposure to a trigger and the capacity of that trigger to elicit a response, is similar for some headache triggers to the pattern observed in anxiety. Short exposure increased the nociceptive response to the trigger but longer exposure decreased the nociceptive response to the trigger. Repeated prolonged exposure was associated with desensitization to the trigger.

6. Behavioral management of headache triggers: coping rather than avoidance

This review has criticized the traditional clinical advice to avoid trigger factors and discussed the anxiety literature that advocates prolonged exposure to anxiety-eliciting stimuli/situations to promote desensitization. The criticisms of the avoidance approach were reinforced by studies showing sensitization and desensitization as a function of length of exposure to empirically-validated headache triggers.

It is argued here that advocating avoidance of headache triggers is too simplistic. It may lead to fewer headaches in the short-term, just as

avoidance of fear-eliciting stimuli may provide transient relief from anxiety. However, such avoidance could lead to more headaches in the long-term, by precluding extinction of the triggers' capacity to elicit headaches, and perhaps even by an insidious sensitization process for some triggers. A more defensible strategy is to advocate 'coping with triggers', construing coping broadly enough to include possible avoidance but also to permit for other coping strategies involving approach/exposure. There are some situations where avoidance of triggers would seem the strategy of choice, but other situations where this would likely be counterproductive.

6.1. Avoidance versus exposure-based strategies

It is not possible within the constraints of this review to exhaustively consider specific exposure-based strategies for every possible type of candidate headache trigger. However, what follows below is a discussion of how this type of approach might apply to some of the most common triggers. This also has the advantage of covering the three main categories of headache triggers – triggers for which an exposure-based approach would seem likely to be the strategy of choice, triggers for which avoidance would seem likely to be the strategy of choice, and triggers for which the choice is likely to be challenging.

6.1.1. Stress, tension and negative emotions

These triggers generally seem good candidates for an exposure-based approach. Headache sufferers need to learn to cope with stress and to be desensitized to situations that elicit maladaptive levels of anxiety. Imaginal exposure therapy has a long history in stress management training dating back at least to the work of Donald Meichenbaum and colleagues on stress inoculation training. It currently is the treatment of choice for a number of stress/anxiety disorders such as Posttraumatic Stress Disorder ([Australian Centre for Posttraumatic Mental Health, 2007](#)). Of course, stressors are too prevalent and diverse to be universally managed through exposure approaches. There may be particularly extreme stressors or anxiety-eliciting situations that can quite effectively be avoided, and that are best avoided, due to the difficulty of learning to cope with them.

6.1.2. Sensory triggers

The sensory triggers of visual disturbance, noise and odours/smells, also seem good candidates for exposure-based approaches, as they are potentially controllable and there is evidence of a relationship between exposure and sensitivity. Once again, though, there are examples of such stimuli for which avoidance would make more sense, such as car exhaust emissions and paint fumes.

6.1.3. Hunger

Hunger is a trigger for which an avoidance approach seems appropriate – encouraging regular meals, having snacks available when long gaps between meals are inevitable, and so forth. Nevertheless, the logic of this review would caution that if this approach is followed too obsessively, there is a danger that individuals become sensitized to hunger, that is, they lose their tolerance for coping with even low levels of hunger. Hence an approach that consists of exposure to mild levels of hunger, with avoidance of more extreme hunger, may be maximally beneficial.

6.1.4. Lack of sleep or excess of sleep

Regular sleep patterns have much to commend them from a healthy lifestyle perspective, just as regular eating habits do. Hence, avoidance of inadequate or excessive sleep is also appropriate. Why excessive sleep should precipitate headaches is not clear, but this may be due to hunger, as sleeping late is often associated with long gaps between eating. As for hunger, an excessive focus on sleep regularity could result in reduced tolerance for deviations in sleep patterns.

Exposure to moderate variability in sleep regularity, coupled with avoidance of extreme variability, may be optimal.

6.1.5. Food and drink

The evidence pertaining to food as a trigger for headaches is equivocal ([Savi et al., 2002](#)), and it is probably true to say that many more people believe that food can trigger their headaches than is actually the case. Indeed, percentage of people for whom food is an important trigger is probably quite low. Nevertheless, it seems worth trying an exposure-based approach with food, as for some this will reveal that the suspected food is not a trigger. For others, exposure may lead to desensitization. Whether or not this is the case may depend on why the food elicits headaches. Where certain foods are clearly established as triggers of headaches, and exposure does not lead to desensitization, then avoidance may become the preferred option. Of course, certain types of food and drink represent special cases, for which other considerations apply. For example, excessive consumption of coffee is considered to have adverse health consequences in addition to the potential capacity of coffee for inducing headaches, and so avoidance of coffee might be more appropriate than attenuating its capacity to induce headaches through systematic exposure. In contrast, drinking plenty of water throughout the day has benefits beyond preventing dehydration as a trigger of headaches.

6.1.6. Alcohol

Alcohol falls into a similar category to food and drink, in that a modest level of exposure may be warranted to test whether indeed it precipitates headaches. Alcohol is a special case, however, in that other considerations must influence decisions concerning alcohol consumption. Avoidance may sometimes be the best approach, especially for individuals at risk of developing dependency and at high-risk times, such as at particular times of the month for females.

6.1.7. Menstruation

Headaches associated with menstruation are considered to arise as a result of low estrogen levels which may be open to manipulation by pharmacological intervention though not behavioral intervention. Such headaches are often associated with other triggers so that if the other triggers can be managed successfully, headaches may not occur. From a behavioral management of headache triggers perspective, the significance of the menstrual cycle may be to serve as a cue to individuals to engage in particular strategies with respect to other triggers, such as avoidance at a particular time of the month.

6.1.8. Weather

Weather as a headache trigger is only controllable to the extent that remaining indoors may isolate an individual from the effects of the weather. If the impact of weather can be controlled in this way, an exposure-based approach could be explored and if it was not successful, avoidance strategies could be employed. There are similarities between weather and menstruation as headache trigger factors, and hence the guidelines for the two triggers have something in common.

6.2. Length and intensity of exposure to triggers

The anxiety literature suggests that prolonged exposure to triggers should be used, with exposure continuing until the anxiety response to the trigger starts to diminish ([Rothbaum & Schwartz, 2002](#)). A direct translation to the headache domain, such that exposure is employed to elicit headaches and is sustained until the headache begins to diminish, probably would not be appropriate. Martin and colleagues have investigated headache triggers in a number of studies and found that once headaches are induced, they are not quick to abate (e.g., [Martin & Teoh, 1999](#); [Martin et al., 2005](#)).

It would seem more appropriate to suggest that one principle of an exposure-based approach to triggers is that exposure should fall short

of inducing a significant headache. The problem is not simply that inducing headaches would be aversive for the patients, as inducing anxiety through exposure to fear stimuli also is aversive in the short-term. Rather the main ground for concern is that there is at least anecdotal evidence amongst clinicians that the more headaches an individual experiences, the more headaches that they are likely to experience in the future. This could occur via a number of mechanisms such as changing the elasticity of the blood vessels that are considered to play a role in headaches, or increased pain sensitivity (Buchgreitz, Lyngberg, Bendtsen, & Jensen, 2006).

It seems plausible that exposure should be long enough to represent a sufficient challenge for learning to take place, whether this learning is best described as desensitization, habituation, extinction, adaptation, developing tolerance, or learning to cope. On the other hand, exposure should fall short of being overwhelming for the patient, or resulting in a severe headache being induced. To use the terminology of Taylor (2002), graduated exposure would be preferable to intense exposure. As the patient adapts/habituates to the trigger, longer exposure could be utilized or exposure to more intense triggers.

7. Conclusions

Chronic headache is a very significant health problem as it is common, causes much suffering, adversely affects an individual's functional capacity, and has a high societal cost. Headaches are precipitated by triggers and advice to avoid triggers is a standard feature of clinical management. However, little research has been carried out on triggers other than to document which triggers are most frequently reported. The headaches by Olesen, Goadsby, Ramadan, Tfelt-Hansen, and Welch (2006), is a large international handbook that is generally regarded as the 'bible' of the field, and includes 139 chapters of which, for example, 16 are devoted to the mechanisms of one type of headache (migraine), but it includes no chapters on triggers. Coverage of triggers is limited to 1 out of 1169 pages.

As the research findings pertaining to headache triggers are limited, a review of this evidence, and consideration of evidence from cognate fields, is useful at this stage for identifying key issues, and suggesting future directions for research, as it would be premature to yet draw definitive conclusions about headache triggers. Nevertheless, some conclusions do already seem justified.

7.1. Triggers and the etiology of headache disorders

The traditional conceptualization of triggers, whether explicit or implicit, is that the capacity to precipitate a headache is an inherent and immutable property of the trigger, and sensitivity to triggers is genetically-determined. This view is not consistent with a number of lines of evidence. Sensitivity, at least to some triggers, is modulated by the environmental factor of exposure history to the trigger.

Fear of the experience of headache drives susceptible individuals to try to avoid headache triggers, and this natural tendency is encouraged by health practitioners and advice on the Internet. Attempts to avoid triggers will result either in no exposure, or probably more commonly short exposure, to the triggers. This may lead to the capacity of the trigger to precipitate headaches being maintained or increasing, through a process of sensitization, failed habituation, or lack of opportunity for learning to cope with the trigger. This suggests that one etiological pathway to developing a chronic headache disorder is attempting to avoid or escape from any stimuli or situation that could trigger a headache.

7.2. Behavioral management of headache triggers

The advice to avoid triggers as a way of preventing headaches is almost certainly counterproductive. Advice to avoid 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. It is likely to lead to fewer headaches in the short-term, and this is probably the reason that this advice continues to be given. Short-term follow-up by health professionals will often elicit the positive feedback from headache sufferers that they have had fewer headaches since adopting this advice. The problem with such advice is that it is likely to lead ultimately to an increase in the potency of some triggers, resulting in more rather than less headaches in the long-term.

This review has argued that the concept of 'coping with triggers' should replace the idea of avoiding triggers. By this it is meant that behavioral management of headache triggers should utilize graded exposure to desensitize headache sufferers to some triggers, whilst reserving avoidance only for selected triggers for which such exposure would seem inappropriate. This approach to headache triggers should result in some trigger factors having a reduced capacity for eliciting headaches, as well as secondary benefits such as a less restricted lifestyle and enhanced self-efficacy for preventing headaches.

7.3. Future research on headache triggers

7.3.1. Investigating triggers

Of the long list of trigger factors that have emerged from studies using retrospective self-report, most have not been investigated using manipulation or even prospective methodologies, leaving open the question of whether such factors really can precipitate headaches. Dietary factors are particularly worthy of investigation, as they are regularly cited as triggers, yet the evidence in support of dietary factors is equivocal. Alternative explanations of the findings are possible. For example, hunger is an established trigger of headaches (Martin & Seneviratne, 1997) and hunger can lead to consuming certain foods. If headaches are experienced following eating those foods, the headaches may be attributed to eating the foods whilst the real trigger may have been the low blood sugar levels that led to the hunger and consequent food consumption.

Research is needed into interactions between triggers as such interactions seem likely, but the only studies to experimentally investigate interactions have failed to find supporting evidence (Martin & Seneviratne, 1997; Martin et al., 2005). The large number of identified headache triggers raises the possibility that triggers may exert their influence in a nested manner, such that a wide array of triggers impact indirectly on headaches through the mediating influence of a smaller number of final trigger pathways, which are directly affected by these first order triggers. For example, triggers A, B and C may all trigger X, and X may trigger a headache; whereas triggers D, E and F all trigger Z, and Z represents an alternative trigger pathway for headaches. Stress and anxiety seem plausible candidates for such final trigger pathways as many triggers are likely to be experienced as stressful (e.g., visual disturbance, noise), and other triggers may elicit anxiety if they have been identified by the sufferer to be precipitants of headaches. A related question worthy of investigation is do different triggers precipitate headaches via the same or different biological pathways?

How do triggers acquire and lose their capacity to precipitate headaches? Behavioral management of triggers should be based on an understanding of factors that increase and decrease the potency of triggers. The anxiety literature provides clues for how to investigate this issue. This review has focussed on length of exposure but other inferences can be drawn from anxiety research. For example, the four pathways to fear identified in the anxiety literature may all have parallels for headaches. With respect to the distinction between 'fears born and bred,' it seems plausible that some triggers have a natural tendency to precipitate headaches, even though this tendency may be modulated by exposure history; whilst the capacity of other triggers to precipitate headaches may be learnt, though such learning may proceed more readily for some trigger factors than for others. Can headache triggers be divided into innate versus learnt triggers? The

most likely candidates for innate triggers are those that are stressful or aversive. Such triggers would include: (i) stress/tension (negative affect); (ii) visual disturbance (flicker, glare and eyestrain); (iii) noise; (iv) hunger; and (v) fatigue. The most likely candidates for learnt headache triggers appear to be the various foods that have been identified as triggers, together with alcohol and smoking. Interestingly, such a dichotomy is in line with the factor analytic study of Martin et al (1993), as the 'innate' triggers listed above loaded on two factors and the 'learnt' triggers on an independent factor. Since the classic work of Garcia and colleagues, we have known that negative associations to food stimuli are very easily conditioned (e.g., Garcia, Ervin, & Koelling, 1966; Garcia & Koelling, 1966). Specifically, strong and lasting aversive reactions can be acquired with ease when the appropriate taste stimulus is associated with illness, even if a long period elapses between tasting the food and the onset of illness. This means that any food eaten before a migraine attack and the onset of nausea is likely to subsequently become a conditioned stimulus, capable of triggering an aversive response that could increase the likelihood of migraine recurring when this food is again consumed.

Anxiety sensitivity, or fear of anxiety, has been shown to be a cognitive vulnerability factor for anxiety disorders (Cox, Borger, & Enns, 1999) and other psychopathology, such as depression (Tull, Gratz, & Lacroce, 2006). The parallel concept of 'fear of pain' has been developed and implicated in the development and maintenance of chronic pain behavior (McCracken, Zayfert, & Gross, 1992). Anxiety sensitivity and fear of pain are related to each other, both are associated with avoidance behaviors (e.g., Martin, McGrath, Brown, & Katz, 2007), and both may operate as risk factors for headache disorders. Parallel scales could be developed measuring 'headache sensitivity' or 'fear of headache', or alternatively, 'headache trigger sensitivity' or 'fear of headache triggers'. Following the logic of the anxiety sensitivity scales, an individual who scored high on 'headache sensitivity' would engage in catastrophic thinking in response to detecting a headache trigger (which could be anxiety) or an early sign of a headache, thus bringing on a headache via increased anxiety. High headache sensitivity would result in attempts to avoid headache triggers, with all the disadvantages that may be associated with this strategy.

The anxiety literature suggests methodologies as well as concepts. For example, the best evidence for vicarious conditioning as a pathway to fear acquisition came from studies that demonstrated vicarious fear-reduction (Rachman, 1977). The distinction between learnt and innate fears largely came from the Dunedin longitudinal study (Poulton & Menzies, 2002). A similar longitudinal study that investigated how the capacity of triggers to precipitate headaches changes or remains constant over time, and what is associated with any changes that occur, would do much to increase our understanding of headache triggers.

7.3.2. Treatment approaches to the triggers of headaches

In the days of 'evidence-based medicine,' it is nothing short of amazing that headaches sufferers around the world are being counseled to avoid trigger factors, when there is so little empirical support for this advice. Randomized controlled trials are needed to evaluate 'avoidance of triggers,' as well as alternative approaches such as the 'coping with triggers' proposed in this review.

The anxiety literature includes many findings that are relevant to using exposure-based techniques for headache triggers. For example, in vivo exposure to anxiety-eliciting situations is superior to exposure to the imaginal presentation of anxiety-eliciting situations (Emmelkamp, 2003). The anxiety literature would suggest that repeated, graduated exposure would be appropriate for 'innate triggers,' while exposure plus a cognitive approach would be most appropriate for 'learnt triggers' (Poulton & Menzies, 2002). Although exposure-based approaches are effective and generally associated with good maintenance, the phenomenon of return of fear to discrete stimuli is a not

uncommon occurrence (Rachman, 1989). Lang, Craske, and Bjork (1999) have suggested a number of specific techniques for the prevention of the return of fear, that may have relevance to preventing the return of the capacity for a trigger to precipitate headaches.

The need for more research on headache triggers is urgent, as clinical practice for many decades has been to encourage avoidance of triggers, an approach that could increase the potency of some triggers resulting in more rather than less headaches in the longer term.

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