

Stress and primary headache: review of the research and clinical management

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Abstract

This review begins with a discussion of the nature of stress and then presents the functional model of primary headache as a framework for conceptualizing the complex relationship between stress and headaches. Research is reviewed on stress as a trigger of headaches, and how stress can play a role in the developmental and psychosocial context of headaches. Clinical management of headaches from a stress perspective is considered both at the level of trials of behavioral interventions that broadly fit into the stress management category, and the additional strategies that might be useful for individual cases based on the research demonstrating associations between stress and headaches. The review concludes by suggesting that although some researchers have questioned whether stress can trigger headaches, overall the literature is still supportive of such a link. Advances in methodology are discussed, the recent emphasis on protective factors is welcomed, and directions for future research suggested.

Introduction

Nature of stress. Stress research has a long history and includes the work of such luminaries as Walter Canon with the ‘fight-flight’ response in the 1930s, and Hans Selye with the General Adaptation Syndrome in the 1950s. Perhaps the modern era of stress research began with the work of Richard Lazarus and his colleagues (most notably Susan Folkman) in the 1980s. Whilst there are many models of stress, most are variations of the transactional model forwarded by Lazarus [1]. Transactional models view psychobiological stress responses as arising from an imbalance between perceived demands and the perceived personal and social resources of the individual to meet the demands [2]. Stress most commonly occurs when perceived demands exceed perceived resources, but can arise when perceived resources exceed perceived demands (e.g., in monotonous work situations where skills are seen as outstripping the demands of the work). The emphasis is on the word ‘perceived’ as it is the ‘subjective’ assessment of demands and resources that determines level of stress, rather than an ‘objective’ assessment.

Research over the years has identified many of the factors that contribute to psychosocial demands such as ‘intensity’ and ‘chronicity’ of the demands. ‘Novelty’, ‘predictability’, ‘complexity’ and ‘control’, also impact on psychosocial demands. Psychosocial resources include ‘appraisal processes’ and ‘psychological coping strategies’. ‘Prior experience’ related to the demands, ‘perceived control’, ‘personality’, and ‘social support’ are also important aspects of psychosocial resources.

An important distinction in the stress literature is between ‘stressful life events’ (also known as ‘major life events’) and ‘minor life events’ (also known as ‘daily hassles’) [3]. Stressful life events range from death of one’s child or being fired from a job to more mundane but still problematic events such as moving house. Daily hassles might include being stuck in a traffic jam, doing household chores, or waiting in a queue. Stress research

has investigated the impact of both major and minor life events on health and wellbeing. It is also important to emphasize that stress researchers have a broader definition of events that can cause stress than do most members of the community. For example, in the Social Readjustment Rating Scale, the events considered to cause most stress would come as little surprise to anyone – ‘death of spouse’, ‘divorce’, and ‘jail term’[4]. But not far below these items are ‘marriage’, ‘marital reconciliation’, and ‘retirement’. Also on the list at a lower level are ‘outstanding personal achievement’, ‘vacation’, and ‘Christmas’. These events are listed because they do require ‘change in life adjustment’, but if clinicians question their patients about ‘stressful life events’, many patients will not think of such events.

Functional model of primary headache. A functional model has been proposed which conceptualizes headaches in terms of their controlling variables, that is, the antecedents and consequences of headaches [5-7]. The model seeks to address questions such as: why does an individual experience a headache at one point in time rather than another point in time; why is the individual experiencing headaches at this time during his/her life rather than at other times; why did the headaches begin when they did or change significantly when they did; and why is the individual vulnerable to experiencing headaches? The immediate antecedents are the factors which precipitate headaches, that is, the triggers. Setting antecedents are the psychosocial context of headaches, that is, the lifestyle factors that moderate current vulnerability. Onset antecedents are the events that resulted in the headaches developing initially or becoming significantly worse. Predisposing antecedents are the constitutional (genetic) and personality characteristics that account for individual differences in vulnerability to headaches. Of the four categories of antecedents, stress is most commonly considered as an immediate antecedent of headaches, but is also relevant to the other three categories. Inadequate social support may be a setting antecedent as it increases vulnerability to stressors. Stressful events may be an onset antecedent if they play a role in the

development of a headache disorder. Vulnerability to stress may be a predisposing antecedent.

The consequences of headaches can be divided into the reactions of the headache sufferers and their significant others to the headache occurring, and the long-term effects of the headache disorder on the headache sufferer and significant others. The etiological significance of these factors lies in the potential for feedback loops that make the headaches worse. Again, stress is relevant to all four categories of consequences. Headaches constitute stressors, and if an individual reacts to headaches with high levels of stress then this can create a vicious cycle of stress-headache-stress. Similarly, if significant others react to headaches in ways that increase the stress levels of the headache sufferer, then feedback loops can be established. With respect to the long-term consequences of having a headache disorder, if having a headache spoils social functions on a number of occasions, this might result in discontinuing attending such events. In turn, this may lead to a reduced social network and less social support, which makes headache sufferers more vulnerable to stress.

Only the antecedent side of the model will be used for structuring the research literature as the significance of the consequences of headaches is the vicious cycles that can develop between the consequences and antecedents. Hence, most research related to the stress-headache relationship could be discussed under antecedents or consequences. For example, low social support is both a contributor to stress-related headaches (that is, an antecedent) and a potential consequence of headaches. It is important to be aware of the consequences side of the model however, when considering the clinical management of headaches from a stress perspective.

Research Literature on the Relationship Between Stress and Headaches

Stress as a trigger of headaches. The evidence for stress as an immediate antecedent or trigger of headaches comes from three main types of studies: (i) retrospective studies (asking

headache sufferers whether stress triggers their headaches); (ii) prospective studies (headache sufferers concurrently self-monitoring stress and headaches); and (iii) laboratory studies (investigating effect of induced stress). A number of recent papers have raised methodological issues with this literature. Lipton and colleagues have published a table of the strengths and limitations of the different types of studies [8]. One of their conclusions was that establishing the relationship between antecedent events and headaches is a “formidable challenge”. Turner and colleagues identified three basic assumptions for determining whether ‘triggers’ cause headaches and argued how difficult it was to evaluate these assumptions [9]. In a companion study, a subset of these researchers examined one of the assumptions, namely the need for ‘constancy in trigger presentation’, and got nine headache sufferers to monitor headaches and three potential triggers over a period of approximately three months [10]. Using sophisticated statistical techniques they concluded that the day-to-day variability was substantial enough for it to be very difficult to draw conclusions about triggers.

Notwithstanding the valid points made in these papers, the results from retrospective studies almost uniformly support stress as the most common trigger in adults both for migraine [11, 12] and for tension-type headache (TTH) [13, 14], and for migraine in children and adolescents [15]. The percentage endorsing stress as a trigger is high in most studies. For example, in the five studies cited, the percentages reporting stress as a trigger range from 71% to 97%. A recent study from Turkey identified stress as the most common trigger of migraine (79% of sample) [16], and a recent study from Brazil reported strong associations between migraine and ‘job stress’, specifically, low job control, high job demands, and low social support [17].

Prospective studies have to confront the issue of the anticipated temporal relationship between stress and headaches. Tunis and Wolff published a model of migraine in which the build up to a migraine took place over three days [18] and this model influenced prospective

studies in the 1980s and early 1990s. For example, one study compared occurrence of stressful events over the four days leading up to and including a migraine day with the occurrence of stressful events over four headache-free days [19]. This study demonstrated significant elevations in stressful events in the four days associated with migraine. Other prospective studies from this period have reported significant associations between stress and headaches but have emphasized individual differences in the magnitude of the stress-headache relationship [20, 21].

One recent large prospective study ($n = 5,159$) investigated stress levels and headache days per month in 3-month blocks across a 2-year period [22]. Stress intensity was associated with headaches for individuals suffering from TTH, migraine and coexisting migraine and TTH, with a stronger relationship in the former group. In TTH sufferers, the relationship between stress and headaches was greater in the younger age group. A prospective study that included physiological measures of stress (saliva cortisol, heart rate average and heart rate variability) as well as perceived stress, investigated stress over 4 days prior to onset of migraine attacks [23]. The study reported the unexpected finding of no significant relationships between the stress variables and headaches. When the data were re-analyzed for the nine participants who believed that $>2/3$ of their migraine attacks were triggered by psychosocial stress, a significant relationship was found between perceived stress and headaches but not between any of the physiological measures of stress and headaches.

The study had a small sample ($n = 17$) of which only nine participants believed that stress was a trigger of their headaches and there is no reason to believe that a relationship between stress and headaches will be found for all migraineurs. Also, the physiological measures of stress used in the study are open to criticism. For example, heart rate is a measure of arousal and can be elevated for reasons other than stress (e.g., climbing stairs or drinking coffee). Salivary cortisol has been used extensively as a biomarker of psychological

stress but is an indirect measure influenced by a number of psychological and biological variables [24]. The authors suggest the possibility of using epinephrine and norepinephrine measured from urine samples in future prospective stress studies [23].

Another recent prospective study investigated reduction in perceived stress as a migraine trigger [25], as there is a literature on ‘let-down’ headaches and ‘weekend’ headaches. The study reported that level of stress was not associated with migraine, but decline in stress from one evening to the next was associated with increased migraine onset over the subsequent 6, 12 and 18 hours. The support for the let-down hypothesis added to a literature that has produced mixed findings. For example, two retrospective studies of samples with migraine and TTH reported that 32-35% of their participants experienced weekend headaches [26, 27]. In contrast, two prospective studies found no support for weekend headaches [28, 29], and in fact one of these studies reported that relaxation after stress had a protective effect against migraine attacks [29]. The authors of the recent study offer a number of explanations for their findings and emphasize the need for more research with a larger sample [25].

It has been suggested that triggers aggregate together to precipitate headaches. One recent study investigated the relationship between stress and sleep duration and headaches in chronic headache sufferers [30]. This study demonstrated that two consecutive days of either high stress or low sleep were strongly predictive of headache, whereas two days of low stress or adequate sleep were protective. Another study investigated stress and menstruation and migraine, and reported that migraine in women was mainly associated with stress, and women are more susceptible to stress in the premenstrual period [31].

With respect to laboratory studies of stress as a trigger, two early studies exposed headache sufferers to mental stress and found that between 63% and 83% reported headaches in response [32, 33]. We have reported three studies in which headache sufferers (migraine and TTH) and control participants were exposed to a stressor (35 minutes of difficult-to-solve

anagrams accompanied by failure feedback) compared to a control condition [34-36]. In each study the stress condition was associated with increased ratings of head pain compared to the control condition. These studies were all completed some years ago but provide some of the strongest evidence that stress can cause headaches as they involve a manipulation of stress.

Stress as a psychosocial and developmental context for headaches. Studies relevant to stress as a setting antecedent factor for headaches include studies of life events, daily hassles, and perceived stress as they relate to headaches. Also relevant are studies of the variables that play an important role in the stress response, namely studies of appraisal, coping and social support. Studies investigating the relationship between stressful life events and headaches have used different measures and reported mixed results. Studies using the Social Readjustment Rating Scale [37, 38] and List of Recent Experiences [39] have found no significant differences between headache sufferers and controls, whilst studies using the Life Events Inventory [40] and Life Experiences Survey [41] have found small but significant differences indicating more stressful life events for headache sufferers. Studies of daily hassles consistently report that headache sufferers experience more hassles than controls [38, 40]. Likewise, studies of perceived stress consistently report higher levels in headache sufferers than controls [42-44].

Holm and colleagues demonstrated that individuals with tension headaches appraise the stressful events they experience more negatively than controls [40]. Specifically, when the potential impact of a stressful event was ambiguous, headache sufferers perceived the event as having a greater impact and themselves as having less control. Also, headache sufferers employed less effective coping strategies in their effort to manage stressful events since they placed more reliance on the relatively ineffective coping strategies of avoidance and self-blame and made less use of social support than did controls. Similarly, Hassinger and colleagues reported that migraineurs responded to stress with wishful thinking, self-criticism,

social withdrawal, and catastrophizing, compared to controls [45]. Najam and Aslam found that for TTH sufferers, as perceived stress increased the use of avoidant coping increased and active coping decreased [46].

A number of studies have found headache sufferers to be at a disadvantage compared to controls on various measures of social support. Martin and Theunissen reported differences between headache and control groups in terms of availability and adequacy of attachment, and availability and adequacy of social integration [39]. Martin and Soon found that headache sufferers were significantly less satisfied with the support available to them and scored lower on all four types of functional support (appraisal, esteem, belonging, and tangible) [42].

Recent studies have yielded findings consistent with earlier studies. Eskin and colleagues reported that patients suffering from TTH and migraine experienced higher perceived stress and deficient social problem solving compared to controls [47]. As mentioned previously, Santos and colleagues found that migraine was associated with low social support [17].

With respect to onset antecedent factors, early studies reported that stress seemed relevant in the onset of headaches in 54% of cases [48], and in the onset of migraine in 50% of cases [49]. More recently, stress has been postulated by a number of authors as a risk factor for headache ‘chronification’ [50-52] but the evidence related to this suggestion is limited. D’Amico and colleagues reported that in their sample, for 44.8% of cases, a stressful event was correlated with the transformation from episodic headache to chronic headache [53]. Minor events (daily hassles) played a greater role in transforming headaches than major events, which the authors interpret as suggesting that patients with transformed headache are characterized by different ways of reacting to stress rather than by greater exposure to major stressful events.

With respect to predisposing antecedent factors, for many years it was argued that headache sufferers tend to have a particular personality profile with descriptions such as

“tense, driving, obsessional perfectionist with an inflexible personality, who maintains a store of bottled-up resentments which can neither be expressed nor resolved” [49, p. 142]. In an early review that tracked down over 100 studies, Blanchard and colleagues concluded that the data do not support the concept of a ‘headache personality’ but that headache sufferers were more psychologically distressed [54]. Other studies have investigated whether particular behavioral styles or patterns are associated with headaches. Studies have reported elevated scores by headache sufferers on two alternative measures of the Type A Behavior Pattern (sometimes referred to as ‘hurry sickness’), the Jenkins Activity Schedule [55, 56] and the measure of Type A from the Framingham study [57]. In a recent study, Hedborg and colleagues found that migraineurs scored high on the personality measure of stress susceptibility [58].

Clinical management of headaches from a stress perspective

Some researchers have developed treatment programs for headaches that are referred to as ‘stress management therapy’/‘stress-coping’ [e.g., 59, 60], but the most common descriptors for behavioral interventions are biofeedback training, relaxation training and cognitive behavior therapy (CBT). The terminology has not been used consistently, however, as biofeedback and CBT often include relaxation training. Some approaches to behavioral treatment of headache incorporate all these techniques [e.g., 61, 62]. Virtually all types of behavioral treatment could be conceptualized as forms of stress management training as, for example: (i) relaxation training is designed to reduce tension; (ii) biofeedback training aims to change physiological variables (e.g., decrease muscle tension) commensurate with lower sympathetic arousal; and (iii) CBT typically targets the dysfunctional thoughts and beliefs associated with stress and negative emotions, as triggers or responses to headaches.

There is now an extensive literature evaluating behavioral treatment for migraine and TTH and there are many reviews [e.g., 63-65], so only a few synoptic comments will be included

here. The efficacy of biofeedback training, relaxation training and CBT is well established. The mechanisms by which they achieve their success is much less clear. In a landmark study of electromyographic biofeedback training, the findings led the authors to argue that the demonstrated reduction in headaches was not a function of reduced activity in the targeted muscle, but resulted from participants viewing their headaches as having a more internal locus of control and themselves as more self-efficacious (i.e., as capable of influencing their headaches), following treatment [66]. These cognitive changes were argued to lead to new and more persistent efforts to cope with headache-related stress that in turn altered the psychobiological stress-response triggering headaches. A number of studies have replicated the finding that behavioral treatment is associated with similar cognitive changes [e.g., 67], and there is evidence that self-efficacy moderates the impact of stressful events on headache [68].

Versions of behavioral treatment that require less therapist time ('minimal therapist contact' – MTC - programs) have been developed and reviewers have concluded that with rare exceptions, the benefits of MTC interventions rivalled those of standard therapist-administered interventions [69-71]. Internet versions of behavioral interventions have been developed, but evaluations of these interventions have produced mixed results. For example, an early study reported a clinically significant reduction in headaches for 50% of users of their internet intervention [72]. However, the drop-out rate in this study was high at 56%. A recent large RCT ($n = 368$) compared an 8-session online treatment program with minimal e-mail support against a wait-list control group and failed to find a significant difference between the groups in terms of reduction in migraine attack frequency [73].

A new development in this literature is to use Mindfulness-Based Stress Reduction with headache sufferers, but so far this has not led to significant decreases across a range of headache measures [74, 75].

The treatment literature evaluates standardized ‘packages’ for the treatment of primary headache but the discussion in this article on the relationship between stress and headaches suggests that a range of techniques could be useful for reducing headaches via stress pathways for particular individuals. The functional model of primary headache includes four types of antecedent factors and four types of consequences, and potential targets for treatment can be found in any of these categories. For example, if a headache sufferer has inadequate social support (setting antecedent) then this could be a target for treatment, or if headaches began as a consequence of sexual abuse (onset antecedent) then this might be an appropriate focus. If a headache sufferer meets the criteria for the Type A Behavior Pattern (predisposing antecedent) then treatment could include techniques designed to change this behavioral style. On the consequences side, if a headache sufferer responds to a headache beginning to develop by trying to do all the tasks that she/he would not be able to do when incapacitated by a headache, then part of the treatment plan might be to change that behavior to a more adaptive reaction. If significant others respond to headaches by expressing anger or frustration, rather than being supportive, then this might be a treatment goal.

With respect to stress as a trigger of headaches, there is a long standing tradition of encouraging headache sufferers to avoid headache triggers. For example, one of the ‘seven elements of good headache management’ of the World Health Organization is “identification of predisposing and/or trigger factors and their avoidance through appropriate lifestyle change” [76, p. 77]. In contrast, we have argued in a series of reviews, on the basis of consideration of cognate literatures and our laboratory research, that avoidance of triggers runs the risk of sensitization to the triggers or reduced tolerance for the triggers [77-79]. We have argued in these reviews for an alternative approach to trigger management called ‘Learning to Cope with Triggers’ (LCT), in which triggers that are inconsistent with good health (e.g., fasting, dehydration, inadequate sleep) should be avoided, but programmed

exposure with the goal of desensitization is the preferable strategy with other triggers (e.g., flicker/glare/eyestrain, noise, anxiety). In a recent RCT, we demonstrated that LCT was associated with approximately three times the reduction in headaches as advice to avoid all triggers [80]. Three cases treated with LCT have recently been published to illustrate the approach [81]. ‘Coping’ with triggers rather than avoidance of triggers is beginning to receive some acceptance in the literature [e.g., 82, 83].

It is clearly unrealistic to advise avoiding ‘stress’, if the term ‘stress’ refers to events (major or minor) that can lead to a psychobiological stress response, as such events are an inescapable fact of life for everyone. The phrase ‘Learning to Cope with Triggers’ seems particularly apt with respect to stress as individuals need to learn to cope with events that can evoke stress. Stress management training typically involves exposure rather than avoidance as participants are taught stress management strategies and then encouraged to practice using these strategies under conditions of mildly elevated stress, often achieved via participants imagining themselves in a stressful situation. In LCT we use such an approach, specifically stress inoculation training [84]. Of course, this is not to suggest that some extreme stressors may best be avoided if that is possible, at least at times of high vulnerability, such as on specific days in the menstrual cycle for females.

Conclusions

Reflecting on the progress that has been made on understanding the relationship between stress and primary headache in the last decade it is easy to be negative about it. For example, a number of researchers have made an eloquent case as to how difficult it is to establish unequivocally that a trigger is a trigger [8-10], and doubts have been cast as to whether stress is a trigger [e.g., 23]. Interpretations of literatures are always a matter of judgement as definitive evidence is rarely available, but it is the opinion of this reviewer that taken overall

the case for stress as a trigger of some headaches in some people is still strong. And the relationship between stress and headaches is not limited to stress as a trigger.

Some of the positives that have come out of the recent literature are methodological advances. Researchers have argued for the advantages of using a causal model to test predictions with respect to whether triggers cause headaches [9]. With respect to measurement, electronic diaries have many advantages over the paper diaries traditionally used for prospective studies of the relationship between stress and headaches [85] and should be used in future research [8]. Suggestions have been made as to physiological measures that could be used in prospective studies [23]. Novel, sophisticated statistical techniques have been utilized for studying the relationship between stress and headaches [e.g., 10, 22].

An interesting development in the literature has been the introduction of protective factors, defined by Lipton and colleagues as “exposures associated with a decreased probability of an attack over a relatively brief period of time” [8]. Knowledge of protective as well as trigger factors has an obvious role to play in headache management but as with trigger factors, the issue of establishing causality is apparent. Some identified protective factors seem likely to have a direct causal effect such as ‘2 days of low stress or adequate sleep’ [30]. However, an event typically experienced as highly stressful could serve as a protective factor if it led to behaviors that reduced stress (e.g., relaxation practice, meditation, massage).

Future research should utilize the methodological advances discussed above, and as some of the contrary findings were in studies with small samples, replicating the research with larger samples would be informative. Topics that seem particularly worthy of further research in this domain include: (i) interactions between stress and other triggers; (ii) the timing between stress levels rising or falling and headache onset, and whether this is affected by the potency of the trigger; (iii) individual differences in the relationship between stress and headaches; (iv) the role of stress in onset of a headache disorder and headache chronification;

and (v) the mechanisms of behavioral interventions (e.g., are the benefits moderated/mediated by changes in stress variables?).

Reference list

1. Lazarus RS, Folkman S: *Stress, Coping and Appraisal*. New York: Springer-Verlag; 1984.
2. Steptoe A: Psychobiological processes in the etiology of disease. In Martin PR. (Ed.). *Handbook of Behavior Therapy and Psychological Science: An Integrative Approach* (pp. 325-347). New York: Pergamon; 1991.
3. Kanner AD, Coyne JC, Schaefer C, Lazarus RS: Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *Journal of Behavioral Medicine* 1981, 4:1-29.
4. Holmes TH, Rahe RH: The Social Readjustment Rating Scale. *Journal of Psychosomatic Research* 1967, 11:213-218.
5. Martin PR, Milech D, Nathan PR: Towards a functional model of chronic headaches: Investigation of antecedents and consequences. *Headache* 1993, 33:461-470.
6. Martin PR: *Psychological Management of Chronic Headaches*. New York: Guilford Press; 1993.
7. Martin PR: Psychological management of the common primary headaches. In ML Caltabiano & LA Ricciardelli (Eds.). *Applied Topics in Health Psychology* (pp. 462-476). Chichester, UK:Wiley-Blackwell; 2013.
8. * * Lipton RB, Pavlovic JM, Haut SR, Grosberg BM, Buse D: Methodological issues in studying trigger factors and premonitory features of migraine. *Headache* 2014, November/December:1661-1669.

This review of trigger factors is a unique contribution in a number of ways including providing definitions of terms that are rarely defined, discussing the strengths and weaknesses of the different methodologies used in the literature, and the issues

involved in assessing trigger factors. It includes a discussion of ‘protective factors’ as well as trigger factors, which is a recent development.

9. * Turner DP, Smitherman TA, Martin VT, Penzien DB, Houle TT: Causality and headache triggers. *Headache* 2013, 53:628-635.

This paper explores the conditions necessary for concluding that ‘triggers’ really do precipitate headaches by applying a causal model from the field of philosophy of science.

10. * Houle TT, Turner DP: Natural experimentation is a challenging method for identifying headache triggers. *Headache* 2013, 53:636-643.

This paper discusses the challenges in determining causal relationships between triggers and headaches, and applies sophisticated statistical techniques to data collected over approximately three months to support their case.

11. Kelman L: The triggers or precipitants of the acute migraine attack. *Cephalalgia* 2007, 27:394-402.

12. Deniz O, Aygul R, Kocak N, Orhan A, Kaya MD: Precipitating factors of migraine attacks in patients with with and without aura. *The Pain Clinic* 2004, 16:451-456.

13. Spierings LH, Ranke AH, Honkoop PC: Precipitating and aggravating factors of migraine versus tension-type headache. *Headache* 2001, 41:554-558.

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

15. Neut D, Fily A, Cuvellier J-C, Vallée L: The prevalence of triggers in paediatric migraine: a questionnaire study in 102 children and adolescents. *Journal of Headache and Pain* 2012, 13:61-65.

16. Mollaoğlu M: Trigger factors in migraine patients. *Journal of Health Psychology* 2012, 18:984-994.
17. * Santos IS, Griep RH, Alves MGM, Goulart AC, Barreto SM Chor D, Bensensor IM: Job stress is associated with migraine in current workers: the Brazilian longitudinal study of adult health (ELSA-Brasil). *European Journal of Pain* 2014, 18:1290-1297.
This study is interesting because it breaks down stress into the variables that contribute to stress, namely low control, high demand, and low social support.
18. Tunis MM, Wolff HG: Studies on headache: Long-term observations of the reactivity of the cranial arteries in subjects with vascular headache of the migraine type. *Archives of Neurology, Psychiatry* 1953, 70:551-557.
19. Levor RM, Cohen MJ, Naliboff BD, McArthur D, Heuser G: Psychosocial precursors and correlates of migraine headache. *Journal of Consulting and Clinical Psychology* 1986, 54:347-353.
20. Kohler T, Haimerl C: Daily stress as a trigger of migraine attacks. Results of thirteen single-subject studies. *Journal of Consulting and Clinical Psychology* 1990, 58:870-872.
21. Mosley TH, Penzien DB, Johnson CA, Brantley PJ, Wittrock DA, Andrew ME, Payne TJ: *Stress and headache: A time-series approach* 1990: Paper presented at the 24th meeting of the Association for Advancement of Behavior Therapy, San Francisco, CA.
22. * Schramm SH, Moebus S, Lehmann N, Galli U, Obermann M, Bock E, Yoon M-S, Diener H-C, Katsarava Z: The association between stress and headache: A longitudinal population-based study. *Cephalalgia* 2015, 35:853-863.

This study is noteworthy more for its impressive methodology (large sample size, data collection over a long period, three headache types diagnosed, sophisticated statistical analysis) than its results, which were consistent with expectations rather than novel.

23. Schoonan GG, Evers DJ, Ballieux BE, Geus EJ, de Kloet ER, Terwindt GM, van Dijk JG, Ferrari MD: Is stress a trigger factor for migraine? *Psychoneuroendocrinology* 2007, 32:532-538.

24. Hellhammer DH, Wüst S, Kudielka BM: Salivary cortisol as a biomarker in stress research. *Psychoneuroendocrinology* 2009, 34:163-171.

25. * Lipton RB, Buse DC, Hall CB, Tennen H, DeFreitas TA, Borkowski TM, Grosberg BM, Haut SR: Reduction in perceived stress as a migraine trigger: Testing the “let-down” hypothesis. *Neurology* 2014, 82:1395-1401.

The finding that a reduction in stress from one day to the next is associated with migraine onset the next day is interesting. The small literature related to this finding is not entirely consistent and a strength of this paper is the different mechanisms suggested to account for the phenomenon and suggestions for future research.

26. Torelli P, Cologno D, Manzoni GC: Weekend headache: A retrospective study in migraine without aura and episodic tension-type headache. *Headache* 1999, 39:11-20.

27. Vaitl D, Propson N, Stark R, Walter B, Schienle A: Headache and sferics. *Headache* 2001, 41:845-853.

28. Morrison DP: Occupational stress in migraine is weekend headache a myth or reality? *Cephalalgia* 1990, 10:189-193.

29. Wöber C, Brannath W, Schmidt K, Kapitan M, Rudel E, Wessely P, Wöber-Bingöl C, and the PAMINA Study Group: Prospective analysis of factors related to migraine attacks: The PAMINA study. *Cephalalgia* 2007, 27:304-314.

30. Houle TT, Butschek RA, Turner DP, Smitherman TA, Rains JC, Penzien DB: Stress and sleep duration predict headache severity in chronic headache sufferers. *Pain* 2012, 153:2432-2440.
31. Parashar R, Bhalla P, Rai NK, Pakhare A, Babbar R: Migraine: Is it related to hormonal disturbances or stress? *International Journal of Women's Health* 2014, 20:921-925.
32. Gannon LR, Haynes SN, Cuevas J, Chavez R: Psychophysiological correlates of induced headaches. *Journal of Behavioral Medicine* 1987, 10:411.
33. Haynes SN, Gannon LR, Bank J, Shelton D, Goodwin J: Cephalic blood flow correlates of induced headaches. *Journal of Behavioral Medicine* 1990, 13:467-480.
34. Martin PR, Seneviratne HM: Effects of food deprivation and a stressor on head pain. *Health Psychology* 1997, 16:310-318.
35. Martin PR, Teoh H-J: Effects of visual stimuli and a stressor on head pain. *Headache* 1999, 39:705-715.
36. Martin PR, Todd J, Reece J: Effects of noise and a stressor on head pain. *Headache* 2005, 45:1353-1364.
37. Andrasik F, Blanchard EB, Arena JG, Teders SJ, Teevan RC, Rodichok LD: Psychological functioning in headache sufferers. *Psychosomatic Medicine* 1982, 44:171-182.
38. De Benedittis G, Lorenzetti A: The role of stressful life events in the persistence of primary headache: major events vs. daily hassles. *Pain* 1992, 51:35-42.
39. Martin PR, Theunissen C: The role of life event stress, coping and social support in chronic headaches. *Headache* 1993, 33:301-306.
40. Holm JE, Holroyd KA, Hursey KG, Penzien DB: The role of stress in recurrent tension headache. *Headache* 1986, 26:160-167.

41. Reynolds DJ, Hovanitz CA: Life event stress and headache frequency revisited. *Headache* 2000, 40:111-118.
42. Martin PR, Soon K: The relationship between perceived stress, social support and chronic headaches. *Headache* 1993, 33:307-314.
43. Yokoyama M, Yokoyama T, Funazu K, Yameshita T, Kondo S, Hosoi H, Yokoyama A, Nakamura H: Association between headache and stress, alcohol drinking, exercise, sleep, and comorbid health conditions in a Japanese population. *Journal of Headache Pain* 2009, 10:177-185.
44. Wacogne C, Lacoste JP, Guilibert E, Hughes FC, Le Jeune C: Stress, anxiety, depression and migraine. *Cephalalgia* 2003, 23:451-455.
45. Hassinger HJ, Semenchuk EM, O'Brien WH: Appraisal and coping responses to pain and stress in migraine headache sufferers. *Journal of Behavioral Medicine* 1999, 22:327-340.
46. Najam N, Aslam S: Perceived stress and coping strategies in headache (migraine and tension-type headache) patients. *Journal of Behavioural Sciences* 2010, 20:1-15.
47. Eskin M, Akyol A, Çelik EY, Gültekin BK: Social problem-solving, perceived stress, depression and life-satisfaction in patients suffering from tension type and migraine headaches. *Scandinavian Journal of Psychology* 2013, 54:337-343.
48. Howarth E: Headache, personality and stress. *British Journal of Psychiatry* 1965, 111:1193-1197.
49. Henryk-Gutt R, Rees WL: Psychological aspects of migraine. *Journal of Psychosomatic Research* 1973, 17:141-153.
50. Bigal ME, Lipton RB: Modifiable risk factors for migraine progression. *Headache* 2006, 46:1334-1343.

51. Scher AI, Midgette LA, Lipton RB: Risk factors for headache chronification. *Headache* 2008, 48:16-25.
52. Houle T, Nash JM: Stress and headache chronification. *Headache* 2008, 48:40-44.
53. D'Amico D, Libro G, Prudenzano MP, Peccarisi C, Guazzelli M, Relja G, Puca F, Genco S, Maggioni F, Nappi G, Verri AP, Cerbo R, Bussone G: Stress and chronic headache. *Journal of Headache Pain* 2000, 1:S49-S52.
54. Blanchard EB, Andrasik F, Arena JG: Personality and chronic headache. *Progress in Experimental Personality Research* 1984, 13:303-360.
55. Hicks RA, Campbell J: Type A-B behavior and self-estimates of the frequency of headaches in college students. *Psychological Reports* 1983, 52:912.
56. Woods PJ, Burns J: Type A behavior and illness in general. *Journal of Behavioral Medicine* 1984, 7:411-415.
57. Martin PR, Nathan PR, Milech D: The Type A Behaviour Pattern and chronic headaches. *Behaviour Change* 1987, 4:33-39.
58. Hedborg K, Anderberg UM, Muhr C: Stress in migraine: personality-dependent vulnerability, life events, and gender are of significance. *Uppsala Journal of Medical Science* 2011, 116:187-199.
59. Holroyd KA, O'Donnell FJ, Stensland M, Lipchik GL, Cordingley GE, Carlson BW: Management of chronic tension-type headache with tricyclic antidepressant medication, stress management therapy, and their combination. A randomized controlled trial. *Journal of the American Medical Association* 2001, 285:2208-2215.
60. Sorbi M, Tellegen B, Du Long, A: Long-term effects of training in relaxation and stress-coping in patients with migraine: A 3-year follow-up. *Headache* 1989, 29:111-121.

61. Holroyd KA, Cottrell CK, O'Donnell FJ, Cordingley GE, Drew JB, Carlson BW, Himawan L: Effect of preventive (β blocker) treatment, behavioural migraine management, or their combination on outcomes of optimised acute treatment in frequent migraine: randomised controlled trial. *British Medical Journal* 2010, 341:c4871.
62. Lipchik GL Holroyd KA & Nash JM: Cognitive-behavioral management of recurrent headache disorders: A minimal-therapist-contact approach. In Turk DC & Gatchel RJ (Eds.) *Psychological approaches to pain management* (2nd ed.). New York: Guilford Press; 2002.
63. Rains JC, Penzien DB, McCrory DC, Gray RN: Behavioral headache treatment: History, review of the empirical literature, and methodological critique. *Headache* 2005, 45 (S2):S92-S109.
64. Andrasik F: What does the evidence show? *Neurological Sciences* 2007, 28: S70-S77.
65. Wells RE, Loder L: Mind/body and behavioral treatments. *Headache* 2012, 52:70-75.
66. Holroyd KA, Penzien DB, Hursey KG, Tobin DL, Rogers L, Holm JE, Marcille PJ, Hall JR, Chila AG: Change mechanisms in EMG biofeedback training: cognitive changes underlying improvements in tension headache. *Journal of Consulting and Clinical Psychology* 1984, 52:1039-1053.
67. Bromberg J, Wood ME, Black RA, Surette DA, Zacharoff KL, Chiauuzzi EJ: A randomized trial of a web-based intervention to improve migraine self-management and coping. *Headache* 2012, 52:244-261.
68. Marlowe N: Self-efficacy moderates the impact of stressful events on headache. *Headache* 1998, 38:662-667.
69. Rowan AB, Andrasik F: Efficacy and cost-effectiveness of minimal therapist contact treatments of chronic headaches: A review. *Behavior Therapy* 1996, 27:207-234.

70. Haddock CK, Rowan AB, Andrasik F, Wilson PG, Talcott GW, Stein RJ: Home-based behavioral treatments for chronic benign headache: A meta-analysis of controlled trials. *Cephalalgia* 1997, 17:113-118.
 71. Swagemakers OTA et al: Behavioural interventions on migraine: The shorter the more effective? *Gedrag en Gezondheid: Tijdschrift voor Psychologie en Gezondheid* 2006, 34:61-74.
 72. Ström L, Pettersson R, Andersson G: A controlled trial of recurrent headache conducted via the internet. *Journal of Consulting and Clinical Psychology* 2000, 68:722-727.
 73. * Kleiboer A, Sorbi M, van Silfhout M, Kooistra L: Short-term effectiveness of an online behavioral training in migraine self-management: A randomized controlled trial. *Behaviour Research and Therapy* 2014, 61:61-69.
- This study reported a negative finding – online treatment not significantly different to a waiting-list control condition. Negative findings are generally considered less exciting than positive findings, but given the large sample size and long experience of the research team, the finding demands attention. Internet therapy is being embraced all over the world due to its use of modern technology, far reach, and low-cost administration, but this study encourages pause for consideration.
74. Schmidt S, Simhauser K, Aickin M, Luking M, Schultz C, Kaube H: Mindfulness-based stress reduction is an effective intervention for patients suffering from migraine – results from a controlled trial. *European Journal of Integrative Medicine* 2010, 2:196.
 75. Cathcart S, Galatis N, Immink M, Proeve M, Petkov J: Brief mindfulness based therapy for chronic tension-type headache: A randomized controlled pilot study. *Behavioural and Cognitive Psychotherapy* 2014, 42:1-15.

76. World Health Organization: *Neurological Disorders: Public Health Challenges*, Geneva, Switzerland: World Health Organization; 2006.
77. Martin PR, MacLeod C: Behavioral management of headache triggers: Avoidance of triggers is an inadequate strategy. *Clinical Psychology Review* 2009, 29:483-495.
78. Martin PR: Managing headache triggers: Think ‘coping’ not ‘avoidance’. *Cephalalgia* 2010, 30:634-637.
79. Martin PR: Behavioral management of migraine headache triggers: Learning to cope with triggers. *Current Pain and Headache Reports* 2010, 14:221-227.
80. * * Martin PR, Callan M, Reece J, MacLeod C, Kaur A, Gregg K, Goadsby PJ: Behavioral management of the triggers of recurrent headache: A randomized controlled trial. *Behaviour Research and Therapy* 2014, 61:1-11.

For decades the standard approach to trigger management has been to counsel headache sufferers to avoid the triggers of their headaches. This study evaluates a new approach which in contrast to the traditional approach includes exposure to some triggers with the goal of desensitizing the triggers. The study demonstrated that the new approach was far more effective than the traditional approach for reducing headaches and medication consumption.
81. Martin PR, Callan M, Kaur A, Gregg K: Behavioral management of headache triggers: Three case examples illustrating a new effective approach (Learning to Cope with Triggers). *Behaviour Change* 2015, 32:202-208.
82. Bendtsen L et al: EFNS guideline on the treatment of tension-type headache – Report of an EFNS task force. *European Journal of Neurology* 2010, 17:1318-1325.
83. Gaul C et al: Team players against headache. *Journal of Head Pain* 2011, 12:511-519.
84. Meichenbaum D: *Stress Inoculation Training*. New York: Pergamon Press; 1985.

85. Richard DCS, Lauterbach D: Computers in the Training and Practice of Behavioral Assessment. In *Comprehensive Handbook of Psychological Assessment. Volume 3*. Edited by Haynes SN, Heiby EB. Hoboken, NJ: John Wiley:222–245; 2004.