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Editorial

Where are we at with stress and headache?

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Background

Chronic Headache (CH) is now in the World Health Organizations top 5 illnesses by disability [1]. Stress is the most commonly reported trigger of a chronic headache episode, however despite over 40 years of research, the mechanisms underlying this are still not clearly understood. The suggestion that pericardial muscles are hyper-reactive to stress in CH sufferers is not supported by the majority of research literature [2]. More recently it has been suggested that CH involves abnormal pain processing in the central nervous system [3]. This raises the possibility of an alternative to the common 'muscle contraction' view of how stress contributes to headache: stress may act via central mechanisms [4] (Figure 1).

Stress and pain are part of an adaptive behavioural system involving the limbic structures, medulla, anterior cingulate cortex (ACC), pre-frontal cortex, front-medial and front-lateral cortices, and soma to-sensory cortices [5]. Chapman et al [5] demonstrate the dynamic interaction of these structures form a system for processing all aversive stimuli. Stress could contribute to pain through this circuitry [5]. Stress has been shown to affect pain processing in the

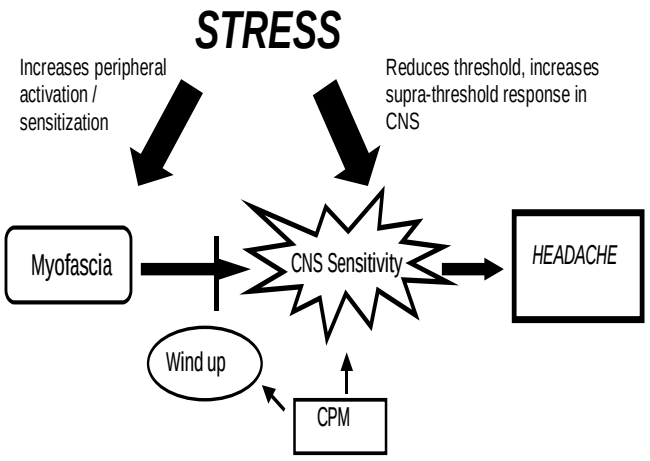

Figure 1: A central model of stress-induced headache. Stress may aggravate sensitization in the pericranial myofascia. Centrally, stress may lower threshold to and increase intensity of noxious signal in the CNS. In CH sufferers, such effects may lower the threshold to, and increase the intensity of, painful input from already tender pericranial musculature, triggering or exacerbating an episode of head pain. Such effects could also contribute to CNS sensitisation, enhanced wind-up, or impaired Conditioned Pain Modulation (CPM) in CH.

hypothalamus, Periaqueductal Grey (PAG), Rostro-ventral Medulla (RVM) and the Locus Coerleus (LC) / noradrenergic (NA) systems [6]. Suzuki et al [7] present evidence for a model of descending facilitatory mechanisms, whereby cortico-limbic activity induced by stress can up-regulate pain, even in the absence of peripheral irritation. Similarly, Stoeter et al [8] suggest cortico-limbic connections common to stress and pain may bind stress-regulating and pain processing systems together, resulting in pain perception being triggered by stress even in the absence of any peripheral noxious input.

Additionally, stress may induce reorganization of primary and secondary cortical nociceptive representations [9], as well as their modulation by fronto-medial, fronto-lateral, and parietal cortex [10]. Jasmin et al [11] demonstrated that changes in gamma-aminobutyric acid (GABA) in the rostral agranular insular cortex (RAIC) can raise or lower pain threshold. Stress, then, could alter the set point of pain threshold in a top down manner via RAIC. A number of healthy human studies found effects of stress on cortical level processing or pain report but no effect on spinal reflexes [12]. Gracely et al [13] and Seminowicz & Davis [14] found that high and low catastrophizers differed on pain sensitivity but not thalamic activation in response to noxious stimulation. Hence, stress may have differential effects on pain signals at spinal and supra-spinal levels in CH sufferers.

Psychologically, stress is proposed by various theories to have facilitatory or inhibitory effects on pain in certain circumstances, through effects on attention and vigilance to pain, arousal attribution, habituation, pain related learning and memory, and reporting behaviour [15]. Stress could therefore aggravate already increased psychological pain processing factors in CH. For example, Stoeter et al [8] suggest central processing of pain and stress may be increased in chronic pain sufferers due to strong memory of previous exposure and enhanced anticipation of new exposure to stress aggravating pain. Supporting this, Armstrong et al [16] reported that headache sufferers reported a greater implicit association between negative events (e.g. stressors) and pain. Stress could also further challenge coping mechanisms, increasing pain reporting and sickness behaviours in CH sufferers [14,17].

Research in headache sufferers

Surprisingly, while stress and hyperalgesia have both been independently associated with headache (see 2,4 for review) very little research has examined their putative interactions in CH sufferers. Cathcart et al reported stress increased pain sensitivity in CH sufferers compared to healthy controls [18]. A further study demonstrated stress-induced headache was associated with stress-induced hyperalgesia in CH sufferers [19]. Interestingly, the same stress task failed to induce headache or increase pain sensitivity in a sample of episodic headache sufferers with normal pain sensitivity (unpublished data in preparation). In another study, self reported pain sensitivity mediated the relationship between daily stress and prospective headache activity in CH sufferers [20]. Stress, pain sensitivity and headache activity were also related to serum levels

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of pro-inflammatory cytokines (submitted). Combined, this initial work supports a central model for stress and headache. Namely, that stress may contribute to CH by aggravating existing pain processing abnormality in CH sufferers [21]. Clearly, these initial findings need replication and extension.

Conclusion

Understanding the importance of stress in CH is important given the high prevalence and cost of headache to society and individual. Stress remains the most commonly reported trigger; however the mechanisms by which it aggravates CH are not well understood. Better understanding of these will aid behavioural and pharmacological intervention for CH.

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