

# Headache Trigger Sensitivity and Avoidance after Mild Traumatic Brain Injury

Noah D. Silverberg,<sup>1</sup> Paul Martin,<sup>2</sup> and William J. Panenka<sup>3</sup>

## Abstract

Most patients with primary headache disorders identify environmental stimuli (e.g., visual glare), situational factors (e.g., stress), physiological states (e.g., hormones), or activities (e.g., exercise) as triggers that elicit or worsen headache episodes. Headache triggers have not been previously studied in post-traumatic headache (PTH). The present study explored the frequency of headache triggers and their avoidance in PTH. Participants with mild traumatic brain injury (TBI) were recruited from outpatient specialty clinics and completed questionnaires at or soon after their initial clinic visit. The research assessment included a list of 12 possible headache triggers where participants were asked to rate sensitivity (how often they get a headache when exposed to the trigger) and avoidance (how often they try to avoid the trigger). Enrolled participants were 40.6 years old (standard deviation [SD]=11.8 years) on average, predominantly female (69.8%) and Caucasian (69.8%), and injured in a motor vehicle crash (47.2%) or fall (20.8%). They were assessed at 9.4 weeks (SD=6.8) post-injury. Most patients with mild TBI in this consecutive series (82.7%) reported ongoing headaches. Mental exertion, stress, and lack of sleep were rated as the most potent headache triggers and among the most frequently avoided. Odors or foods, hunger, and weather conditions were rated as the least potent triggers. Headache trigger sensitivity and avoidance were moderately correlated ( $r=0.736$ ,  $p<0.001$ ). Headache severity was more associated with trigger sensitivity [ $F(2,49)=13.45$ ,  $p<0.001$ ] than trigger avoidance [ $F(2,47)=2.97$ ,  $p=0.062$ ]. In summary, the pattern of headache triggers in persistent PTH after mild TBI appears somewhat different from that in primary headache disorders, with mental exertion emerging as uniquely important. Pervasive avoidance of mental exertion to prevent headaches (cogniphobia) might be a worthwhile behavioral intervention target after mild TBI.

**Keywords:** brain concussion; craniocerebral trauma; headache; post-concussion syndrome; post-traumatic headache

## Introduction

HEADACHE EPISODES ARE OFTEN PRECEDED BY WHAT patients come to identify as triggers, most commonly stress, poor sleep, weather changes, light flicker or glare, hormonal factors, certain foods, and odors.<sup>1,2</sup> It is clinical convention to advise headache sufferers to avoid headache triggers.<sup>3</sup> However, continually avoiding such triggers may be maladaptive because 1) many triggers, such as stress, are ubiquitous and attempting to avoid them would result in an extremely restricted lifestyle, 2) patients are prone to misidentify triggers by erroneously assuming causation between events that occur in close temporal proximity, and 3) avoidance increases sensitivity to some triggers such that over time, they more easily elicit headache.<sup>3–5</sup> Martin and colleagues have reported experimental evidence for the maladaptive role of trigger avoidance<sup>3</sup> and developed a behavioral treatment that aims to improve headaches by reducing avoidance of certain triggers.<sup>5,6</sup>

Headache triggers have not been previously studied in the context of post-traumatic headache (PTH).<sup>7</sup> Headache is among the most common persistent symptoms after traumatic brain injury (TBI).<sup>8–11</sup> Up to 25–45% of patients continue to experience headaches 12 months after injury.<sup>8,11–14</sup> PTH incidence is not associated with TBI severity<sup>11,12</sup> and typically shares clinical features with primary headache disorders (such as migraine or tension-type) and headaches precipitated by traumatic injuries not involving the head.<sup>9,15,16</sup> Clinical practice guidelines recommend that treatment of PTH be guided by the primary headache disorder it most closely resembles.<sup>17,18</sup>

The present study is an initial investigation of headache triggers in PTH. We explore whether the same triggers associated with primary headache disorders are also seen in PTH and whether their avoidance might contribute to chronic PTH. We report on a cross-sectional sample of patients who presented to an outpatient specialty clinic for persistent symptoms after mild TBI, the most

<sup>1</sup>Division of Physical Medicine and Rehabilitation, University of British Columbia, Rehabilitation Research Program, GF Strong Rehab Centre, Vancouver, British Columbia, Canada.

<sup>2</sup>Research School of Psychology, Australian National University, Canberra, Australia.

<sup>3</sup>British Columbia Neuropsychiatry Program, Department of Psychiatry, University of British Columbia, Vancouver, British Columbia, Canada.

common type of TBI. We hypothesized that patients with PTH would identify similar and different triggers in comparison to the primary headache population. Specifically, we expected that 1) the most common triggers in the primary headache disorder literature such as stress and poor sleep would be among the most common triggers in our sample, and 2) additional triggers scarcely recognized in the primary headache disorder literature, such as mental exertion (thinking too hard), would be commonly reported in our sample, based on prior evidence that mental exertion after mild TBI tends to worsen post-concussion symptoms in general<sup>19,20</sup> and particularly headache.<sup>21</sup> We further hypothesized that trigger avoidance would be associated with headache severity and trigger sensitivity. Clarifying headache trigger frequency, sensitivity, and avoidance in a mild TBI sample can help establish the rationale for exposure-based treatment<sup>5,6</sup> and highlight potential protocol modifications for this clinical population.

## Methods

### Participants

The sample for the present study is an amalgamation of patients from two studies. Study 1 is a prospective cohort study that recruited from four outpatient concussion clinics, described in prior publications.<sup>22,23</sup> The headache triggers scale was administered to the last 17 participants who enrolled in this study (August 2016 to February 2017). The headache triggers scale also was administered to the first 40 patients who enrolled in an ongoing randomized controlled trial (ClinicalTrials.gov Identifier NCT03221218; July to December 2017) that recruited from two of the same concussion clinics (Study 2). A diagnosis of mild TBI was an inclusion criterion in both studies. The injury had to occur within the past 6 months for Study 1 and 3 months for Study 2. In Study 1, we confirmed self-reported diagnosis with a structured interview for acute signs and symptoms based on the World Health Organization Neurotrauma Task Force diagnostic criteria.<sup>24</sup> For Study 2, we required that the referring physician documented a diagnosis of mild TBI. Patients were excluded from Study 1 if they were not working at the time of their injury and from Study 2 if they did not have a family physician, consistent with study-specific research aims. Recruitment procedures were similar for both studies. Patients were approached at the first clinic visit and invited to participate in research. Eligible, consenting patients completed an initial in-person assessment that included the questionnaires described below. A sample size of 55 was expected to achieve  $\pm 10\%$  precision in proportion estimates (trigger endorsement), with 90% confidence.

### Measures

**Headaches and post-concussion symptoms.** Participants completed a standardized measure of post-concussion symptoms. Those from Study 1 completed the British Columbia Postconcussion Symptom Inventory (BC-PSI)<sup>25,26</sup> and participants from Study 2 completed the Rivermead Postconcussion Symptom Questionnaire (RPQ).<sup>27,28</sup> Both scales include a single item querying headaches. On the BC-PSI, participants separately rate the frequency (0–5) and severity (0–5) of their symptoms. Frequency and severity ratings are then multiplied for each item. Product scores of 2–6 indicate a mild symptom and product scores of 7 or higher indicate a moderate-to-severe symptom. On the RPQ, an item rating of 2 out of 4 indicates a mild symptom and a rating of 3–4 indicates a moderate-to-severe symptom. Participants who scored  $< 2$  on the headache item of the RPQ or BC-PSI were assigned to the “none” group for analyses.

**Headache Triggers Scale.** A scale was designed for the present study, as no validated self-report measure of headache triggers had been published at the time data collection began. Participants were asked to consider a list of 12 possible headache triggers, and for each, rate on a scale from 1 (never) to 5 (always) how often they get a headache when exposed to the trigger and how often they try to avoid the trigger, yielding measures of trigger sensitivity and trigger avoidance, respectively. Each score could range from 12 to 60. In the present sample, Cronbach's alpha (internal consistency) was 0.82 for the headache sensitivity subscale and 0.82 for the headache avoidance subscale. The Headache Triggers Scale is reproduced in the Appendix. This questionnaire was administered to all participants, whether they reported having recent bothersome headaches (i.e., RPQ item score or BC-PSI item product score of 2+) or not.

### Analysis

Frequency distributions of item endorsement on the Headache Triggers Scale informed our primary hypothesis. The subsamples comprising the total sample were compared with t-tests (for continuous variables) and chi-squared tests (for proportions) on demographic characteristics, injury variables, and outcome measures. Pearson correlation coefficients were used to examine associations between continuous (normally distributed) variables. We performed an analysis of covariance (ANCOVA) with headache severity category (none vs. mild vs. moderate-severe) as the fixed factor, time since injury (in weeks) as a continuous covariate, and headache trigger sensitivity as the dependent variable. A second ANCOVA was run with headache trigger avoidance as the dependent variable. Time since injury was included as a covariate because preliminary analyses revealed that 1) the subsamples differed on this variable, and 2) time since injury correlated with an outcome of interest. Missing data was handled with listwise deletion ( $n < 5$  cases per analysis).

### Results

Sample characteristics are summarized in Table 1. The combined sample was predominantly Caucasian and female, with at least some post-secondary education. Most were injured in a motor vehicle crash or fall, and presented to clinic 2–3 months post-injury. The constituent subsamples (from Study 1 and Study 2) were similar with respect age, gender, ethnicity, and education level. However, participants from Study 2 were assessed significantly sooner, consistent with the more stringent time since injury eligibility criterion. Potentially as a result, participants from Study 2 had more moderate-severe (vs. mild) headaches and reported greater overall headache trigger avoidance than participants from Study 1.

The frequency of headache trigger endorsement in the combined sample is shown in Figure 1. The most potent perceived headache triggers were mental exertion, stress, lack of sleep, and computer screens (mean ratings out of 5 shown in Fig. 1A). Head movement and physical exertion were moderate triggers. Odors or foods, hunger, and weather conditions were least likely to trigger headache (mean [M]  $< 2.0$ ). The most avoided triggers (Fig. 1B) were lack of sleep, stress, loud noise, bright lights, head or neck movement, and mental exertion. Participants rarely ( $M < 2.0$ ) avoided certain foods or weather conditions.

Headache trigger sensitivity and avoidance subscales were moderately correlated ( $r = 0.736$ ,  $p < 0.001$ ). Correlations at the individual item (trigger) level revealed that the sensitivity-avoidance relationship was stronger from some triggers (e.g.,  $r = 0.796$  for head/neck movement and  $r = 0.797$  for odors) than others ( $r = 0.308$  for stress).

TABLE 1. PARTICIPANT CHARACTERISTICS

	Combined sample (n = 53 <sup>a</sup> )	Study 1 (n = 17)	Study 2 (n = 36 <sup>a</sup> )
Age, M (SD)	40.6 (11.8)	43.8 (11.2)	39.1 (12.0)
Gender, n (%) female	37 (69.8%)	11 (64.7%)	26 (72.2%)
Ethnicity, n (%) Caucasian	37 (69.8%)	13 (76.5%)	24 (66.7%)
Education level, n (%) with college diploma	32 (64.2%)	12 (70.5%)	22 (61.1%)
Mechanism of injury, n (%)			
Motor vehicle crash	25 (47.2%)	5 (29.4%)	20 (55.6%)
Fall	11 (20.8%)	6 (35.3%)	5 (13.9%)
Sport	8 (15.1%)	2 (11.8%)	6 (16.7%)
Other	9 (17.0%)	4 (23.5%)	5 (13.9%)
Time from injury to assessment, M (SD) weeks	9.4 (6.8) <sup>b</sup>	16.8 (5.9)	5.9 (3.7)
Access to financial compensation for injury, n (%)	40 (75.5%)	15 (88.2%)	25 (69.4%)
Post-concussion symptom severity, M (SD)			
British Columbia Postconcussion Symptom Inventory	–	21.7 (10.3)	–
Rivermead Postconcussion Symptom Questionnaire	–	–	35.6 (13.5)
Headaches <sup>c</sup> , n (%) endorsed			
None	9 (17.3%)	4 (23.5%)	5 (14.3%)
Mild	8 (15.4%) <sup>d</sup>	6 (35.3%)	2 (5.7%)
Moderate-to-severe	35 (67.3%) <sup>d</sup>	7 (41.2%)	28 (80.0%)
Headache Triggers Scale, M (SD)			
Sensitivity	33.7 (8.3)	31.4 (8.4)	34.9 (8.0)
Avoidance	33.9 (8.4) <sup>d</sup>	29.3 (8.4)	36.3 (7.5)

<sup>a</sup>Two participants were missing data on the Headache Triggers Scale.

<sup>b</sup>Study groups different based on t-test  $p < 0.05$ .

<sup>c</sup>Based on the headache item from the British Columbia Postconcussion Symptom Inventory for participants in Study 1 (0–1 = none, 2–6 = mild,  $\geq 7$  = moderate-severe) and from the Rivermead Postconcussion Symptom Questionnaire for participants in Study 2 (0–1 = none, 2 = mild, 3–4 = moderate-severe).

<sup>d</sup>Study groups different based on chi-squared test  $p < 0.05$ .

M, mean ; SD, standard deviation.

Time since injury was negatively correlated with trigger avoidance ( $r = -0.302$ ,  $p = 0.037$ ) but not trigger sensitivity ( $r = -0.096$ ,  $p = 0.506$ ). Controlling for time since injury, headache severity was more associated with trigger sensitivity [ $F(2,49) = 13.45$ ,  $p < .001$ ] than trigger avoidance [ $F(2,47) = 2.97$ ,  $p = 0.062$ ]. The mean score on the headache trigger sensitivity subscale was 37.24 ( $SD = 5.54$ ) for participants with moderate-severe headaches ( $n = 33$ ), 29.00 ( $SD = 8.30$ ) for participants with mild headaches ( $n = 8$ ), and 24.63 ( $SD = 9.09$ ) for participants who reported experiencing no recent bothersome headaches ( $n = 8$ ).

## Discussion

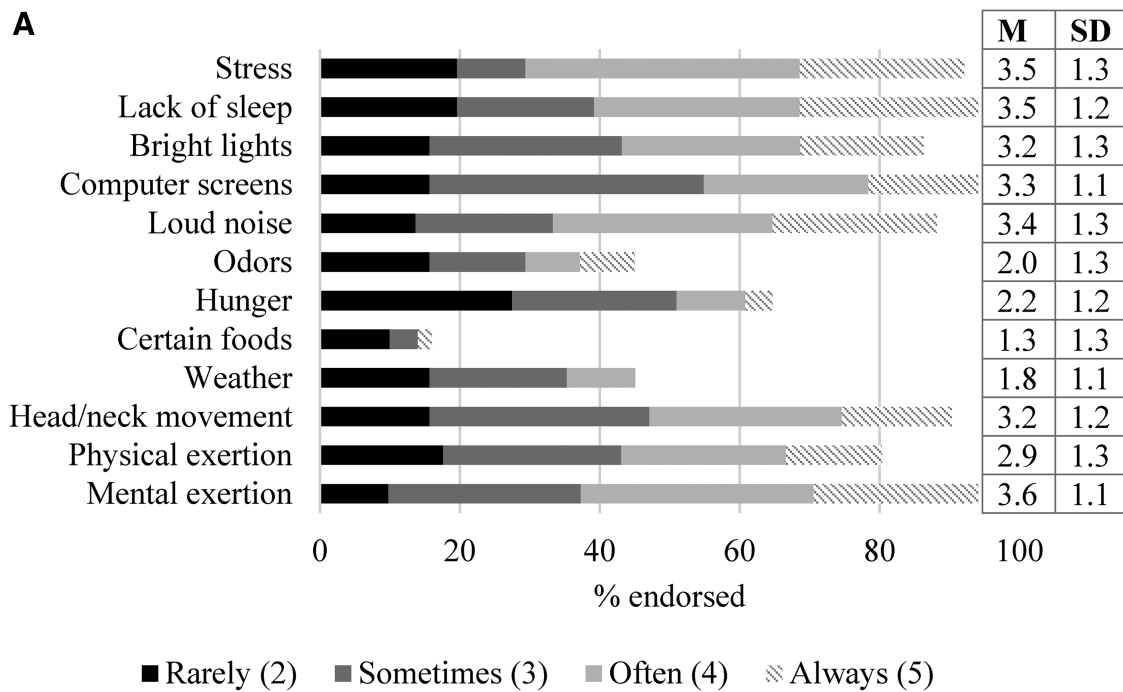
Participants with mild TBI perceived mental exertion, stress, and lack of sleep to be the most potent headache triggers. Stress and poor sleep were similarly found to be the most common headache triggers in a recent meta-analysis of primary headache disorder studies.<sup>1</sup> However, mental exertion was largely absent from this body of literature. It is unclear if mental exertion is an atypical trigger in primary headache disorders (i.e., is relatively unique to PTH) or whether it has merely received insufficient attention. The closely related concept of “cogniphobia” originated in a PTH context.<sup>29</sup> Cogniphobia has since been considered in primary headache disorders,<sup>30,31</sup> but it is not known how commonly mental exertion is perceived as a headache trigger in comparison to other headache triggers in this population.

It is possible that mental exertion provokes psychological stress, which in turn elicits a headache. In other words, stress could be an important mechanism by which mental exertion becomes a headache trigger. Mental exertion may be particularly stress-provoking for people with persistent symptoms after mild TBI to the extent

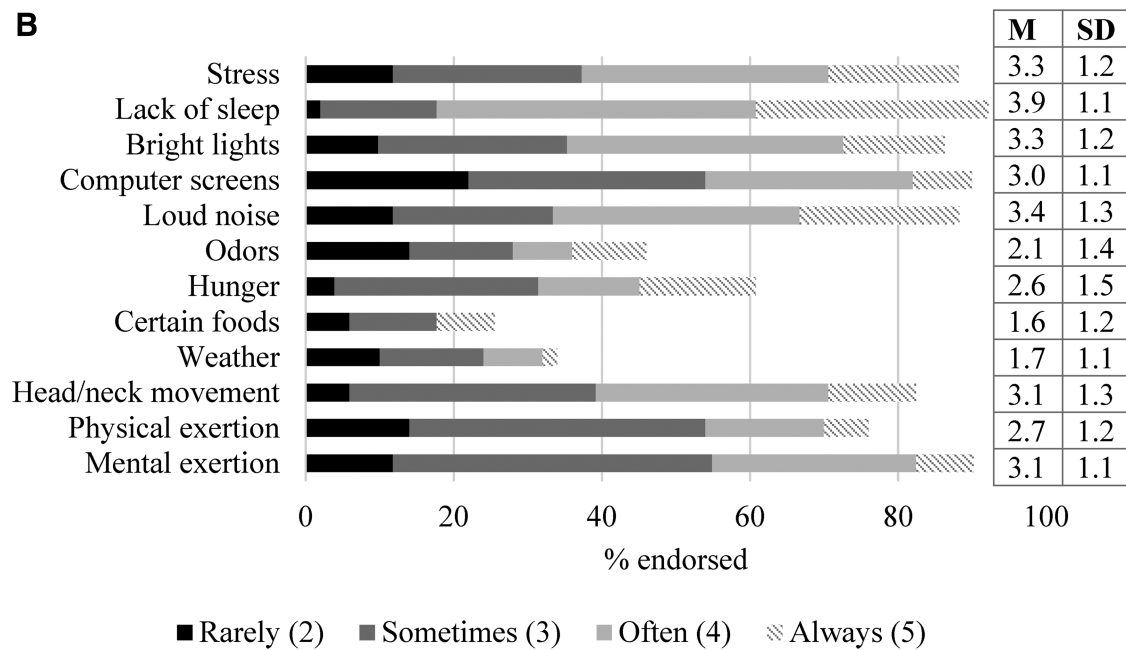
they hold negative self-evaluative beliefs about their cognitive abilities.<sup>32</sup> When there is an imbalance between one’s perceived resources (impaired cognitive abilities) and demands (a cognitively challenging task), stress may arise.<sup>33</sup> Although most eligible studies in the Pellegrino and colleagues meta-analysis<sup>1</sup> reporting on “stress” as a trigger queried negative emotions and/or difficult life circumstances, one defined stress as mental effort and another as prolonged concentration. Further research will be necessary to understand whether mental exertion is a distinct headache trigger with distinct biological mechanisms (e.g., cerebrovascular dysfunction),<sup>7,34</sup> or merely a common situational stressor after mild TBI.

Head/neck movement and computer screens also were perceived to be potent headache triggers in our mild TBI sample. Vestibular injury and/or cervical strain co-occurring with mild TBI may explain the aversiveness of head/neck movement. Photophobia and/or other visual disturbances after mild TBI may contribute to intolerance of computer screens. Odors, hunger, foods, and weather conditions are among the most studied triggers in the primary headache disorder literature<sup>1,2</sup> but were perceived as least potent in our sample. These triggers may be less relevant to PTH.

The present study yielded other novel findings. First, not surprisingly, headache triggers perceived as more potent tended to be avoided more. However, this relationship was stronger for triggers that could more readily be avoided (e.g., odors) than triggers that are relatively unavoidable (e.g., stress). This observation warrants further investigation. Second, participants who endorsed more frequent/severe headaches perceived being more sensitive to headache triggers. The relationship between headache frequency/severity and trigger avoidance was much weaker. This pattern suggests that patient characteristics other than their headache (e.g., personality, premorbid coping style, illness beliefs) may motivate avoidance



**FIG. 1A.** Trigger sensitivity.



**FIG. 1B.** Trigger avoidance.

and/or that avoiding triggers is not a highly effective PTH management strategy.

Several limitations of the present study should be highlighted. First, participants were pooled from two studies with similar but not identical eligibility criteria and methodologies. Participants were all recruited from the same clinics, in the same manner. The post-concussion symptom scales used in each study have been shown elsewhere to correlate strongly (0.78).<sup>25</sup> Participants from each sample differed on time since injury; we statistically controlled for time since injury in pooled analyses. Second, headache triggers avoidance was measured with a new questionnaire that has not undergone rigorous psychometric development and external validation. Since data collection for the present study began, Kubik and Martin published on the Headache Triggers Sensitivity and Avoidance Questionnaire in a primary headache sample, demonstrating strong internal consistency, test-retest reliability, and construct validity.<sup>35</sup> Future studies should weigh the brevity of the scale used in the present study with the comprehensiveness and known psychometric characteristics of Kubik and Martin's questionnaire.<sup>35</sup> TBI researchers who choose the latter should consider adding an item for mental exertion.

Third, using a questionnaire method likely affected how many triggers were reported. Participants tend to report fewer triggers when queried with an open-ended interview question.<sup>1</sup> This also has been shown for post-concussion symptom reporting.<sup>36</sup> Fourth, all data for the present study were collected at a single point in time, making it impossible to draw conclusions about causality. Our findings are consistent with experimental evidence<sup>37,38</sup> and theoretical models<sup>3</sup> that assume avoidance of headache triggers results in sensitization to triggers, justifying exposure-based treatment. Although avoiding certain triggers might heighten their potency, avoidance may not necessarily increase headache frequency, so long as people continue to avoid those triggers. Fifth, headache phenotypes were not characterized in this sample. Based on prior research, migraine is the most common type of post-traumatic headache (40–60%), followed by tension-type (30–50%) and cervicogenic (10–20%).<sup>8,39,40</sup> Differences in trigger profiles between headache phenotypes appears modest.<sup>1,2</sup> Nevertheless, this warrants further study. Sixth, the majority of participants were receiving or seeking some form of financial compensation for their injury. There is a risk that a general over-reporting response style contributed to the high observed rates of headache trigger endorsement. Finally, some participants may have had daily headaches that predated their TBI, and so would not be strictly classified as “PTH” cases. The Headache Triggers Scale does not explicitly ask respondents to focus on their current (post-injury) headaches. It is possible that when completing this questionnaire in the context of a TBI research study, some participants may have considered their experience with headache triggers prior to the injury.

Management of persistent PTH is challenging. There are few evidence-based treatment options, and incomplete treatment response with conventional therapies is common.<sup>40</sup> Cohort studies suggest that a chronic, intractable course is common.<sup>8,41</sup> Improved characterization of causal and maintaining factors could direct clinical trialists to investigate promising new therapies. For example, to the extent that trigger avoidance perpetuates PTH, a behavioral intervention that teaches patients how to more effectively manage triggers that are not readily avoidable<sup>6</sup> should be prioritized for research. The present study suggests that when applying behavioral treatment protocols developed for primary headache disorders<sup>5,6</sup> to patients with mild TBI, mental exertion, head/neck movement, and computer screen use should be incorporated.

In summary, trigger avoidance is very common in primary headaches,<sup>1–3</sup> but to our knowledge, this is the first study to investigate headache triggers and their avoidance in a TBI sample. Study participants endorsed a variety of headache triggers, with a pattern that differed somewhat from that seen in primary headache disorders. Notably, mental exertion emerged as the most potent headache trigger in our sample, suggesting that this trigger may be relatively unique to PTH sufferers. Avoiding mental exertion to prevent headaches (cogniphobia) may be associated with reduced cognitive performance<sup>21,30</sup> and other forms of avoidant coping,<sup>21</sup> which in turn, might contribute to chronic disability after mild TBI.

## Acknowledgments

This study was funded by a Specific Priorities Research Grant from WorkSafeBC (#RS2014-SP03). NDS receives salary support from a Health Professional Investigator Award from the Michael Smith Foundation for Health Research.

## Author Disclosure Statement

Noah Silverberg has a private practice in neuropsychology that includes consultation roles with professional sport organizations and disability insurance providers, including WorkSafeBC.

For the other authors, no competing financial interests exist.

## References

1. Pellegrino, A.B.W., Davis-Martin, R.E., Houle, T.T., Turner, D.P., and Smitherman, T.A. (2018). Perceived triggers of primary headache disorders: a meta-analysis. *Cephalalgia* 38, 1188–1198.
2. Turner, D.P. and Houle, T.T. (2017). Influences on headache trigger beliefs and perceptions. *Cephalalgia* 38, 1545–1553.
3. Martin, P.R. and MacLeod, C. (2009). Behavioral management of headache triggers: Avoidance of triggers is an inadequate strategy. *Clin. Psychol. Rev.* 29, 483–495.
4. Martin, P. (2000). Headache triggers: to avoid or not to avoid, that is the question. *Psychol. Health* 15, 801–809.
5. Martin, P.R. (2010). Behavioral management of migraine headache triggers: learning to cope with triggers. *Curr. Pain Headache Rep.* 14, 221–227.
6. Martin, P.R., Reece, J., Callan, M., MacLeod, C., Kaur, A., Gregg, K., and Goadsby, P.J. (2014). Behavioral management of the triggers of recurrent headache: a randomized controlled trial. *Behav. Res. Ther.* 61, 1–11.
7. Kamins, J. and Charles, A. (2018). Posttraumatic headache: basic mechanisms and therapeutic targets. *Headache* 58, 811–826.
8. Lucas, S., Hoffman, J.M., Bell, K.R., and Dikmen, S. (2014). A prospective study of prevalence and characterization of headache following mild traumatic brain injury. *Cephalalgia* 34, 93–102.
9. Lucas, S. (2015). Posttraumatic headache: clinical characterization and management. *Curr. Pain Headache Rep.* 19.
10. Levin, H.S., Mattis, S., Ruff, R.M., Eisenberg, H.M., Marshall, L.F., Tabaddor, K., High Jr., W.M., and Frankowski, R.F. (1987). Neurobehavioral outcome following minor head injury: a three-center study. *J. Neurosurg.* 66, 234–243.
11. Dikmen, S., Machamer, J., Fann, J.R., and Temkin, N.R. (2010). Rates of symptom reporting following traumatic brain injury. *J. Int. Neuropsychol. Soc.* 16, 401–411.
12. Hoffman, J.M., Lucas, S., Dikmen, S., Braden, C.A., Brown, A.W., Brunner, R., Diaz-Arrastia, R., Walker, W.C., Watanabe, T.K., and Bell, K.R. (2011). Natural history of headache after traumatic brain injury. *J. Neurotrauma* 28, 1719–1725.
13. Faux, S. and Sheedy, J. (2008). A prospective controlled study in the prevalence of posttraumatic headache following mild traumatic brain injury. *Pain Med.* 9, 1001–1011.
14. Kraus, J., Hsu, P., Schaffer, K., Vaca, F., Ayers, K., Kennedy, F., and Afifi, A.A. (2009). Preinjury factors and 3-month outcomes following emergency department diagnosis of mild traumatic brain injury. *J. Head Trauma Rehabil.* 24, 344–354.

15. Seifert, T. and Evans, R. (2010). Post-traumatic headache: a review. *Curr. Pain Headache Rep.* 14, 292–298.
16. Lucas, S., and Ahn, A.H. (2018). Posttraumatic headache: classification by symptom-based clinical profiles. *Headache J. Head Face Pain* 58, 873–882.
17. Department of Veterans Affairs/Department of Defense. (2016). VA/DoD Clinical Practice Guidelines/ Management of Concussion-mild Traumatic Brain Injury (mTBI). [www.healthquality.va.gov/guidelines/Rehab/mtbi](http://www.healthquality.va.gov/guidelines/Rehab/mtbi) (Last accessed December 22, 2018).
18. Ontario Neurotrauma Foundation. (2018). Guidelines for Concussion/Mild Traumatic Brain Injury and Persistent Symptoms, 3rd Edition, for Adults over 18 Years of Age. <https://braininjuryguidelines.org/concussion> (Last accessed December 22, 2018).
19. Silverberg, N.D., Iverson, G.L., McCrea, M., Apps, J.N., Hammeke, T.A., and Thomas, D.G. (2016). Activity-related symptom exacerbations after pediatric concussion. *JAMA Pediatr.* 170, 946–953.
20. McLeod, T.C.V., and Gioia, G.A. (2010). Cognitive rest: the often neglected aspect of concussion management. *Hum. Kinet. Athl. Ther.* Today 15, 3.
21. Silverberg, N.D., Iverson, G.L., and Panenka, W.J. (2017). Cogniphobia in mild traumatic brain injury. *J. Neurotrauma* 34, 2141–2146.
22. Silverberg, N.D., Panenka, W.J., and Iverson, G.L. (2017). Work productivity loss after mild traumatic brain injury. *Arch. Phys. Med. Rehabil.* 99, 250–256.
23. Silverberg, N.D., Panenka, W., and Iverson, G.L. (2018). Fear avoidance and clinical outcomes from mild traumatic brain injury. *J. Neurotrauma* 35, 1864–1873.
24. Carroll, L.J., Cassidy, J.D., Holm, L., Kraus, J., and Coronado, V.G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on mild traumatic brain injury. *J. Rehabil. Med.* 36, 113–125.
25. Sullivan, K. and Garden, N. (2011). A comparison of the psychometric properties of 4 postconcussion syndrome measures in a nonclinical sample. *J. Head Trauma Rehabil.* 26, 170–176.
26. Iverson, G.L., Lovell, M.R., and Collins, M.W. (2003). Interpreting change on IMPACT following sport concussion. *Clin. Neuropsychol.* 17, 460–467.
27. King, N.S., Crawford, S., Wenden, F.J., Moss, N.E.G., and Wade, D.T. (1995). The Rivermead Post Concussion Symptoms Questionnaire: a measure of symptoms commonly experienced after head injury and its reliability. *J. Neurol.* 242, 587–592.
28. Potter, S., Leigh, E., Wade, D., and Fleminger, S. (2006). The Rivermead Post Concussion Symptoms Questionnaire: a confirmatory factor analysis. *J. Neurol.* 253, 1603–1614.
29. Martelli, M.F., MacMillan, P., and Grayson, R. (1999). Kinesiophobia and cogniphobia: avoidance-conditioned pain-related disability (ACPRD). *Arch. Clin. Neuropsychol.* 14, 804.
30. Suhr, J. and Spickard, B. (2012). Pain-related fear is associated with cognitive task avoidance: exploration of the cogniphobia construct in a recurrent headache sample. *Clin. Neuropsychol.* 26, 1128–1141.
31. Seng, E.K. and Klepper, J.E. (2017). Development of the cogniphobia scale for headache disorders (CS-HD): a pilot study. *Psychol. Assess.* 29, 1296–1301.
32. Potter, S. and Brown, R.G. (2012). Cognitive behavioural therapy and persistent post-concussional symptoms: integrating conceptual issues and practical aspects in treatment. *Neuropsychol. Rehabil.* 22, 1–25.
33. Lazarus, R. (1966). Psychological stress and the coping process. McGraw-Hill: New York.
34. Tan, C.O., Iverson, G.L., and Taylor, J.A. (2014). Cerebrovascular regulation, exercise, and mild traumatic brain injury. *Neurology* 83, 1665–1672.
35. Kubik, S.U. and Martin, P.R. (2017). The Headache Triggers Sensitivity and Avoidance Questionnaire: establishing the psychometric properties of the questionnaire. *Headache* 57, 236–254.
36. Nolin, P., Villemure, R., and Heroux, L. (2006). Determining long-term symptoms following mild traumatic brain injury: method of interview affects self-report. *Brain Inj.* 20, 1147–1154.
37. Martin, P.R. (2001). How do trigger factors acquire the capacity to precipitate headaches? *Behav. Res. Ther.* 39, 545–554.
38. Martin, P.R., Lae, L., and Reece, J. (2007). Stress as a trigger for headaches: relationship between exposure and sensitivity. *Anxiety Stress Coping* 20, 393–407.
39. Lucas, S., Hoffman, J.M., Bell, K.R., Walker, W., and Dikmen, S. (2012). Characterization of headache after traumatic brain injury. *Cephalalgia* 32, 600–606.
40. Brown, A.W., Watanabe, T.K., Hoffman, J.M., Bell, K.R., Lucas, S., and Dikmen, S. (2015). Headache after traumatic brain injury: a national survey of clinical practices and treatment approaches. *PM R* 7, 3–8.
41. Lucas, S., Smith, B.M., Temkin, N., Bell, K.R., Dikmen, S., and Hoffman, J.M. (2016). Comorbidity of headache and depression after mild traumatic brain injury. *Headache* 56, 323–330.

Address correspondence to:

Noah D. Silverberg, PhD  
 Rehabilitation Research Program  
 GF Strong Rehab Centre  
 4255 Laurel Street  
 Vancouver, British Columbia V5Z 2G9  
 Canada

E-mail: noah.silverberg@vch.ca

## APPENDIX: HEADACHE TRIGGERS SCALE

**Instructions:** Some people notice that their headaches have triggers. For example, you might do, see, hear, smell, or feel something and then develop a headache, or experience a worsening of your headache. For each possible trigger listed below, please rate how strong the trigger is for you, ranging from never (I haven't notice that this trigger affects my headaches at all) to always (I get a headache every time I am exposed to this trigger). Naturally, people tend to avoid headache triggers in order to get fewer headaches. Please also rate how much you try to avoid each trigger, from never (I don't try to avoid it) to always (I avoid it as much as I can). There is space at the bottom to write in triggers you have noticed that are not listed here.

Possible Trigger	How often do you to get headache (or experience a worsening in your headache) after this trigger?					How much do you try to avoid this trigger to manage your headaches?				
	Never	Sometimes		Always		Never	Sometimes		Always	
1 Stress	1	2	3	4	5	1	2	3	4	5
2 Lack of sleep	1	2	3	4	5	1	2	3	4	5
3 Bright lights	1	2	3	4	5	1	2	3	4	5
4 Computer screens	1	2	3	4	5	1	2	3	4	5
5 Loud noise	1	2	3	4	5	1	2	3	4	5
6 Odors	1	2	3	4	5	1	2	3	4	5
7 Hunger	1	2	3	4	5	1	2	3	4	5
8 Certain foods	1	2	3	4	5	1	2	3	4	5
9 Weather	1	2	3	4	5	1	2	3	4	5
10 Head or neck movement	1	2	3	4	5	1	2	3	4	5
11 Physical exercise	1	2	3	4	5	1	2	3	4	5
12 Mental exertion (e.g., thinking or concentrating too hard or too long)	1	2	3	4	5	1	2	3	4	5