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How Does the Migraine Attack Stop? It's NOT the trigger: Common Headache triggers do not Predict Cessation of Pain

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We have investigated the headache-eliciting effect of a wide range of “triggers” or precipitants that either are directly perceived (e.g., stress, specific foods, sleep) or are less readily perceptible (e.g., hormonal fluctuations, barometric changes, autonomic arousal). In so doing, we have created mathematical models to predict the probability that an individual will experience an attack when preceded by various combinations of potential triggers. An interesting question that can be asked with these models but that to date remains unaddressed is whether or not the forces that trigger an attack might also play a role in stopping one. To address this question, we have reversed our model to predict the flip-side of the more usual trigger question. In other words, instead of inquiring “does the headache trigger (or absence thereof) predict onset of an attack?”, we are inquiring “does the headache trigger (or absence thereof) predict the cessation of the attack?” The evidence so far rejects that common headache triggers predict the cessation of an attack.

To illustrate this inquiry, we examined N = 9 episodic headache sufferers who participated in a diary study where they recorded headache activity using diaries as well as perceived stress using the daily stress inventory daily for one month.[1,2] Stress is widely considered to be one of the most potent triggers of headache, but is it also predictive of attack cessation?[3] To examine the question, we coded the duration between each headache attack as well as the duration of each attack, with individuals contributing multiple attacks to the analysis. For the purposes of graphical display and analysis, we artificially dichotomized each day's stress rating as either being “high” (above that participant's mean ratings during the month) or “low” (below that participant's mean ratings). Then, we conducted a survival analysis examining the course of headache activity for either: a) days after an attack had just ceased, or b) days after an attack had just started. In this way, we examined the impact of stress on either eliciting a new attack or ceasing an existing attack.

Figure A displays the time-course of days between attacks. On day 1 of this plot, participants did not experience a headache and were dichotomized into groups experiencing either “high” or “low” stress. After this day, the proportion of days that they were still free from headache was plotted using a Kaplan-Meier curve. As can be seen from the curves, when participants experienced “high” stress, they were much more likely (HR: 2.6, 95% CI: 1.29 to 5.4) to experience a new-onset headache in each of the following days. The plot supports the popular wisdom that stressful days can elicit an attack.

Disclosures

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Figure B displays the duration of an attack. On the first day of the attack (day 1), participants again were dichotomized into groups experiencing either “high” or “low” stress. After this day, the proportion of days where a headache is still experienced is plotted using a Kaplan-Meier curve. The role of stress in predicting the end of the attack is much less clear than when initiating it. On subsequent days after participants experienced “high” stress, they were somewhat, yet not statistically more likely to experience the cessation of the attack (HR: 1.7, 95% CI:0.86 to 3.5). This modest association is certainly counterintuitive and would be difficult to explain because stress has been linked to increased pain reporting in rheumatoid arthritis and other chronic pain states. [4]Taken together there is not evidence that stress can serve substantially to “trigger” attack cessation the way it appears to trigger initiation.

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Kaplan-Meier (survival) plots of the role of stress in initiating (A) and ceasing (B) a headache attack. In both plots stress was artificially dichotomized into “high” and “low” levels with the proportion of the sample days that were free from headache (A) or exhibiting a headache (B) plotted versus time since the experience of the stress (day 0).