Introduction

Pain catastrophizing is associated with enhanced pain and also reduces the efficacy of pain inhibitory processes. Interestingly, prior work suggests pain catastrophizing does not exert its hypertalassic effects via descending facilitation of spinal nociception, because several studies have failed to find a relation with the nociceptive flexion reflex (NFR, a physiological marker of spinal nociception). The present study further explored this issue by examining the relationship between situation-specific (state) pain catastrophizing and conditioned pain modulation (CPM). To determine whether state catastrophizing is associated with disrupted conditioned pain modulation, a measure of central pain inhibition.

Methods: Pain Catastrophizing Scale

- Participants completed the Pain Catastrophizing Scale (PCS)
- This scale was altered to represent state/situation specific pain catastrophizing
- Item self-report measure for use in clinical and non-clinical samples; used for persons with and without pain
- Subscales
  - Rumination: (e.g., I can't stop thinking about the pain)
  - Magnification: (e.g., I worry that the pain will get worse)
  - Helplessness: (e.g., There's nothing I can do)
- Pain catastrophizing groups were categorized low, medium, and high using a tertile split

Outcome: Noxious Flexion Reflex (NFR)

- NFR: Spinally-mediated withdrawal reflex elicited by Aδ fibers
- NFR Magnitude: size of the reflex correlates with pain ratings and used for within-subject changes in spinal nociception
- Calculated: d:score (d = mean EMG of 90 to 150 ms stimulation interval) minus mean EMG of -60 to 0 ms pre-stimulation interval) divided by the average SD of -60 to 0 ms pre-stimulation and SD of 90 to 150 ms post-stimulation intervals

Funding Source

Research reported was supported by the National Institute On Minority Health And Disparities of the National Institutes of Health under Award Number R01MD007807. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Conclusions

These findings imply that central modulatory mechanisms are altered by greater pain catastrophizing, shifting the balance toward descending facilitation during CPM and a generalized perceptual hyperalgesia. As such, individuals that catastrophize may be at risk for chronic pain. Our results suggest that while catastrophizing does not affect CPM related inhibition of pain, it may affect descending modulation of spinal nociception via conditioned pain modulation mechanisms.