Does race moderate the relationship between trauma exposure and endogenous pain modulation? Preliminary findings from the Oklahoma Study of Native American Pain Risk (OK-SNAP)


Department of Psychology, The University of Tulsa, 800 South Tucker Drive, Tulsa, OK 74104

Introduction

Native Americans (NA) report a high prevalence of chronic pain, however the mechanisms for this are unknown. Trauma exposure could contribute because trauma survivors also have a higher risk for chronic pain. Conditioned pain modulation (CPM), a measure of endogenous pain modulation in which pain inhibition prevents pain and decreases pain sensitivity, can be related to several chronic pain conditions. Further, deficits in CPM have been noted in some minorities at risk for chronic pain, however the mechanisms for this are unknown. Trauma exposure could contribute because trauma survivors also have a higher risk for chronic pain than the general population. It was hypothesized that elevated trauma exposure is related to reduced CPM and that this relationship is stronger in NAs.

Life Events Checklist and Trauma Demographics

Participants

- 174 healthy, pain-free men and women
  - Characteristics: 76 White (43.68%), 89 Non-Hispanic White/CAucasian (51.15%), 85 Native Americans (43.10%)
  - Average Age = 29.6 yrs (SD=12.93)
  - Married (19%)  
  - Average Amount of Education = Partial College (45.5%)
  - Employed (67.7%)  
  - Exclusion criteria: < 18 years of age; BMI > 35

Procedure

- Overview, Informed Consent & Eligibility Determination (Health Status Screening)
- Two testing sessions were completed on separate days
- Native American heritage was corroborated by a CDIB (certificate degree of Indian blood) card or tribal
- Cardiovascular, neurological, and/or circulatory problems
- Recent use of antidepressant, anxiolytic, antihypertensive medications

Life Events Checklist Administered

- No more than 20 traumatic events in the past 12 months

Outcome: Measurement of NFR

- Noxious Cutaneous Reflex (NFR): a spinal-mediated withdrawal reflex elicited by Aδ fiber activation
- NFR magnitude: Biceps femoris EMG activity in the 90-150 ms post-stimulation window
- Calculated d' score [d' = (mean EMG of 90 to 150 ms post-stimulation interval) minus (mean EMG of 60 to 90 ms pre-stimulation interval) divided by the average SD of EMG from 60 to 90 ms pre-stimulation and SD of 80 to 150 ms post-stimulation intervals]

Outcome: Conditioned Pain Modulation (CPM)

- Conditioned Pain Modulation (CPM): Pain ratings made following each stimulation (verbally for CPM, on a 0–100 numerical rating scale)

Data Analysis

- Mixed ANOVA (pain as a DV and CPM as the IV) conducted in PROCESS for mediation

Conclusions

- Given that NFR is a marker of spinal nociceptive control, these results suggest that trauma may disrupt descending inhibitory circuits that regulate pain signaling at the spinal level
- Further longitudinal research is needed to investigate the role of trauma exposure and race on future development of chronic pain

Funding Source

Research was supported by the National Institute on Minority Health and Health Disparities of the National Institutes of Health under Award Number R01MD007807 and by the National Science Foundation under Award Number 1265718. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health or the National Science Foundation

Life Events Checklist and Trauma Demographics

- Trauma Demographics: Participants completed the Life Events Checklist (LEC) for the DAV-IV
- Self-report measure assessing the number and severity of events a participant has been exposed to
- Multiple items can be endorsed by the participant personally
- Items that happened to the participant personally were summed
- 33.7% of participants endorsed at least 1 trauma

Trauma Endorsement Demographics

- Trauma Type
  - Sample N [%] of Reported Trauma
  - Accident or Environmental Disaster (e.g., Natural disaster, Interpersonal Violence)
  - Physical Assault
  - Combat or Captivity (e.g., Combat or exposure to a war zone)
  - Threatened Death Or Serious Injury (e.g., Life-threatening illness or injury)

Results

- The overall model is significant (F=5.68, p<.02)

- The model predicting CPM of pain was non-significant (R²=.01, p=.91) from trauma exposure
- Neither the race predictor (b=.05, p=.91, CI=[-.01, .08]), nor the trauma exposure predictor (b=.07, p=.14, CI=[-.08, .21]) were significant
- The interaction was also non-significant (p=.02, p=.44, .40)

- Fractional cuts in CPM of pain

- The main effect of race was non-significant (b=-.03, p=.12, CI=[-.07, .01]), nor was the interaction (b=-.01, p=.74, CI=[-.02, .03])

- Outliers were winsorized

- Multiple linear regression (dependent variable: NFR magnitude; independent variables: race, trauma exposure, race x trauma exposure)

- The model predicting CPM of NFR was non-significant (R²=.01, p=.92) from trauma exposure

- Neither the race predictor (b=.05, p=.91, CI=[-.02, .01]), nor the trauma exposure predictor (b=.07, p=.14, CI=[-.08, .21]) were significant
- The interaction was also non-significant (p=.02, p=.44, .40)

- Given that NFR is a marker of spinal nociception, these results suggest that trauma may disrupt descending inhibitory circuits that regulate pain signaling at the spinal level
- This was dependent upon race and indicates that this relationship between trauma and CPM of NFR is similar across racial groups and does not represent a unique pain risk for Native Americans
- Further longitudinal research is needed to investigate the role of trauma exposure and race on future development of chronic pain

Outcomes

- Caution should be taken when interpreting CPM due to the high prevalence of chronic pain in NAs