Is conditioned pain modulation disrupted in sexual assault survivors?


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

Introduction

Sexual assault (SA) is defined as any form of sexual contact that occurs without the explicit consent of the recipient and ranges from unwanted touch to rape. SA is associated with increased chronic pain risk and numerous chronic pain conditions. Recent findings from our research group suggest that SA survivors exhibit hyperalgesia and difficulty engaging in descending modulation of spinal nociception (assessed via the nociceptive flexion reflex (NFR) via electrical processes. To our knowledge, no study has examined the relationship between SA and conditioned pain modulation (CPM; pain inhibits pain). The present study examines the relationship between SA and CPM in a sample of SA survivors and a comparison group of 32 no-SA survivors.

Objective

To assess endogenated pain modulation in 32 SA survivors and a comparison group of 32 no-SA survivors.

Participants

- 32 SA survivors and 32 no-SA survivors sampled from a larger study investigating pain processing in healthy, pain-free Native American and Non-Hispanic White individuals.
- Groups were matched on age, race, sex, and number of no-SA trauma exposures.

Sample Characteristics:
- 55 women
- Average age = 30.74 yrs (SD = 12.29)
- White/Caucasian non-Hispanic (29.4%)
- Married (20.6%)
- Average amount of education = Partial College (42.6%)
- Employed full time (27.9%)

Exclusion criteria:
- < 18 years of age
- BMI > 35
- Current acute illness, psychotic symptoms, chronic pain condition, or inability to speak/read English
- Cardiovascular, neurological, and/or circulatory problems
- Recent use of anesthetic, antidepressant, anxiolytic, antihypertensive medications

Procedure

- Overview, Informed Consent & Eligibility Determination (Health Status Screening)
- Test sessions were counterbalanced and tasks within days were randomized
- Two testing sessions were completed on separate days

- Overview of Procedure

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<thead>
<tr>
<th>Conditioned Pain Modulation (CPM)</th>
<th>Outcome: Measurement of NFR</th>
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</thead>
<tbody>
<tr>
<td>Stimulating electrode over sural nerve</td>
<td>Biceps femoris EMG</td>
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<td>NFR change = mean NFR - baseline NFR</td>
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Life Events Checklist and Trauma Demographics

- Trauma Demographics: Participants completed the Life Events Checklist (LED) for the DSM IV Questionnaire

- Self-report measure assessing the number of potentially traumatic events to which a participant has been exposed

- SA group assignment was based upon if the participant endorsed either of the two SA items “happened to me”

- NFR magnitude: Biceps femoris EMG activity in the 90-150 ms post-stimulus window

- No-SA group did not exhibit CPM of pain whereas the control group did not experience any change in NFR

- Interaction effect for CPM phase by SA history (p = .01)

- Participants provided informed consent after the procedures were explained

- Pre-immersion Water Immersion Post-immersion

Results – CPM of Pain

- Groups did not differ on our surrogate stimulus intensity or NFR threshold (the intensity needed to elicit the NFR)

- Stimulus intensity was entered as a covariate to account for the individually calibrated intensity used during the CPM procedure

- Main effect of group (p = .27)

- Main effect of CPM phase (p = .01)

- Reporting significant inhibition of pain

- Interaction effect for CPM phase by SA history (p = .01), such that SA history did not affect CPM of pain

Results – CPM of NFR Magnitude

- Main effect of group (p = .27)

- Main effect of CPM phase (p ≤ .00)

- Interaction effect for CPM phase by SA history (p = .00)

- Participants provided informed consent after the procedures were explained

- Exclusion criteria:

Data Analysis

- Outliers on all variables were addressed through the use of modified one-step M-estimator according to Hinklay.

- Participant trials with NFR baselines higher than 3 µV were excluded from analyses due to questionable data validity

- 2 Multilevel Models (MLM) for the CPM models with Group and CPM phase as the IVs, and pain ratings and NFR change as DVs

Conclusions

- Modulation of pain perception was intact for both the SA and no-SA comparison group

- The findings of modulation of NFR suggest that SA survivors may display facilitation of spinal nociception

- These findings suggest that inhibitory mechanisms specific to descending circuitry are disrupted in SA survivors

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