Impact of Adolescent Inter-Personal Violence Exposure on Physiological Stress Response and PTSD Symptoms in Adulthood

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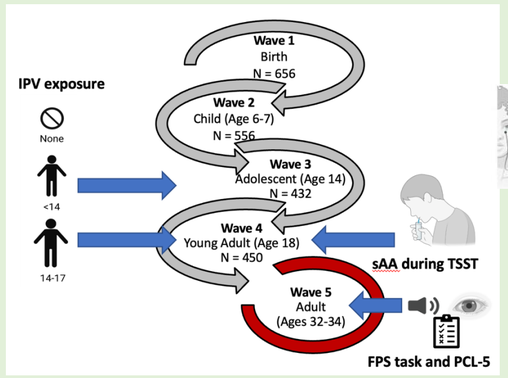
DETROIT TRAUMA PROJECT

BACKGROUND

- Exposure to interpersonal violence (IPV) is associated with increased risk for post-traumatic stress disorder (PTSD) and long-term changes in stress response systems[1].
- Despite research on both IPV and PTSD, it is not clear how developmental timing of IPV contributes to dysregulated physiological fear responses in adulthood.
- Fear-potentiated startle (FPS) in the presence of safety signals is impaired in individuals with elevated PTSD symptoms, which may be associated with heightened sympathetic nervous system arousal[2].
- Salivary alpha-amylase (sAA) is a biomarker of sympathetic nervous system activation and an index of physiological stress responses[3].
- **Study Aim:** To examine longitudinal associations among IPV exposure, stress reactivity (sAA) at age 18, and FPS to safety cues and PTSD symptoms in adulthood.

METHODS

- 105 participants were recruited from a longitudinal birth cohort study (SchooBE study). Participants were grouped based on self-reported IPV exposure: **no IPV, IPV before 14 years and IPV between 14 and 18 years.**
- **Age 18: sAA** was collected during the **Trier Social Stress Test (TSST).**
- **Age 32-34:** Participants completed a differential fear conditioning **FPS task**, using electromyogram of eyeblink startle response to a threat (CS+) and a safety cue (CS-). The PTSD Symptom Checklist for DSM-5 (PCL-5) was also completed.



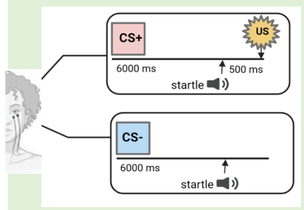
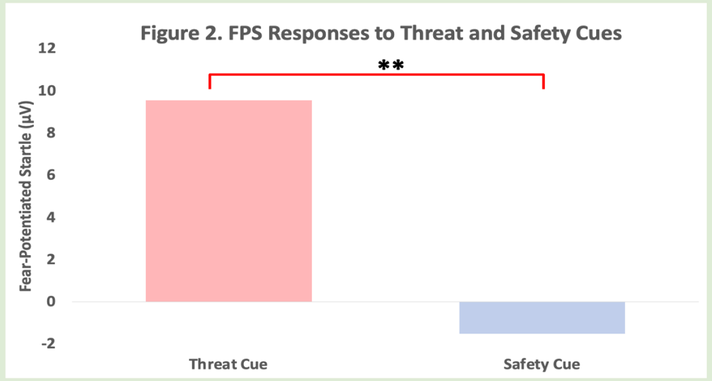
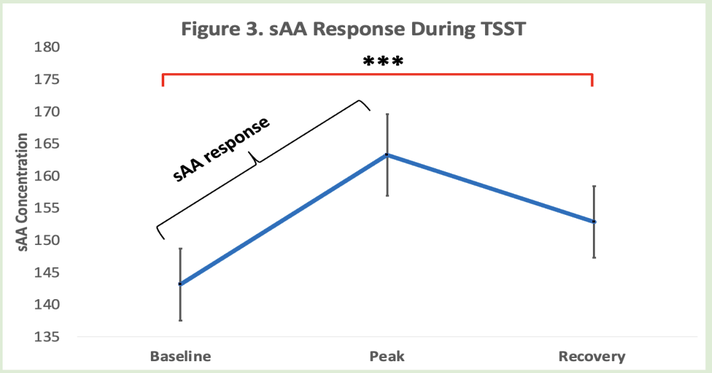


Figure 1. A. Study waves and data collection B. Fear-potentiated startle paradigm

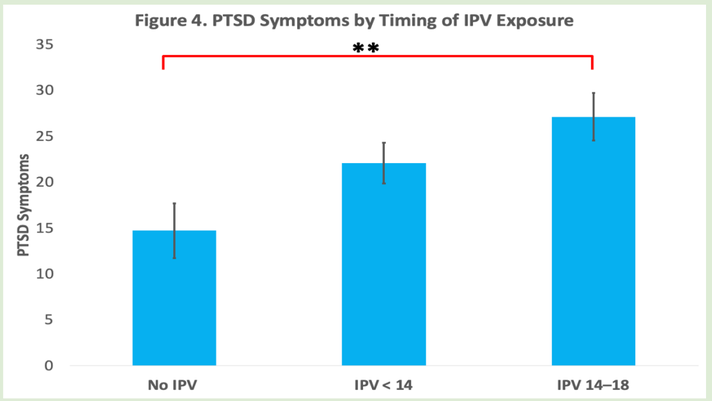
RESULTS



A main effect of trial type indicated greater response to the threat than safety cue ($F(1,44)=8.42$, $p<.006$).



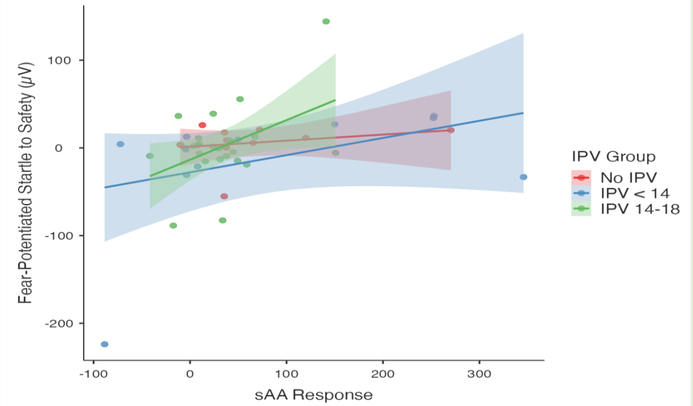
sAA differed over time ($F(2,770)=16.13$, $p<.001$) with Peak > Recovery > Baseline (all $p \leq .036$).



**$p<.01$; ***$p<.001$

There was a significant IPV group difference in adulthood PTSD symptoms ($F=4.91$, $p=.009$), where participants exposed to IPV between the ages of 14 and 18 showed higher symptom scores compared to unexposed peers.

Figure 5. Relationship Between sAA Response and Safety Cue Startle Responses by Timing of IPV Exposure



- There were no main effects of IPV exposure groups in sAA levels at age 18 or adulthood FPS response to safety cues.
- Within the age 14-18 IPV group, higher sAA reactivity to the TSST at age 18 was significantly associated with greater FPS response to safety cues in adulthood ($r=.443$, $p=0.039$)
- Association between FPS to safety cues and PTSD symptoms was not significant, suggesting that other resilience factors (e.g., family support, emotional intelligence) may modulate or buffer long-term outcomes.

CONCLUSIONS

- Adolescence may represent a sensitive window of development during which IPV exposure particularly increases PTSD risk.
- Within the adolescent IPV exposure group, greater sympathetic reactivity at age 18 (sAA) was associated with heightened fear responses to safety cues in adulthood, indicating potential dysregulation in fear inhibition processes.
- Protective factors may influence whether dysregulated fear responses translate into clinical symptoms.

REFERENCES

1. Chen Y., Shen Q., Lichtenstein P., et al. (2024) Incidence Trajectories of Psychiatric Disorders After Assault, Injury, and Bereavement. *JAMA Psychiatry* 81(4):374–385. https://doi:10.1001/jamapsychiatry.2023.5156
2. Jovanovic T., Norrholm SD., Blanding NQ., et al. (2010) Impaired fear inhibition is a biomarker of PTSD but not depression. *Depress Anxiety*. 27(3):244-51. https://doi.org/10.1002/da.20663
3. Thoma, M. V., Kirschbaum, C., Wolf, J. M., & Rohleder, N. (2012). Acute stress responses in salivary alpha-amylase predict increases of plasma norepinephrine. *Biological psychology*, *91*(3), 342–348. https://doi.org/10.1016/j.biopsycho.2012.07.008

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