Sex-specific association between high traumatic stress exposure and social cognitive functioning in youth

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Abstract

Background: Traumatic Stressful Events (TSE) during childhood and adolescence are associated with increased risk for psychopathology and with cognitive impairment. Aberrations in social cognition may contribute to the psychopathology risk. We examined performance differences on social cognitive measures between youth with high TSE exposure and no TSE exposure, and how these effects vary in females and males.

Methods: The Philadelphia Neurodevelopmental Cohort (PNC) investigates clinical and cognitive phenotypes in a US youth (aged 8–21) community population. Here we compare performance in social cognition tasks between youth with high exposure (3 TSE, N=830) and youth with no exposure (N=5202). Three social cognition tasks were analyzed: (1) Age Differentiation, (2) Emotion Identification (happy, sad, angry, fearful, or neutral) and (3) Emotion Intensity Differentiation (happy, sad, angry, and fearful).

Results: A significant TSE Group by Sex interaction was observed in all social cognitive tasks. In the Emotion Identification task, males with high traumatic stress exposure out-performed non-exposed males; exposure did not impact performance in females. In the Emotion Intensity Differentiation task, females with high traumatic stress exposure performed worse than non-exposed females, with no difference in males between exposure groups. Exploratory analyses

revealed sex differences were driven by improved identification of angry expressions in stress exposed males and poorer performance in differentiating intensity of happy expressions in stress exposed females.

Conclusion: Exposure to high levels of early life traumatic stress was associated with sex-specific differences in social cognition. These findings might be related to the sex specific patterns of psychopathology emerging during adolescence.

Keywords

Traumatic Stress; Social Cognition; Developmental Psychopathology; Sex Differences; Childhood Trauma; Emotion Processing

Introduction

The association between traumatic or stressful events during childhood and the subsequent development of psychopathology is well established (1, 2). Mounting evidence suggests that, in clinical psychiatric populations, exposure to traumatic stress early in life has detrimental developmental effects that are often associated with a more severe course of illness (3–5) and decreased treatment response (6– 9). Despite the well-established link between trauma and psychopathology, more research is needed to elucidate the mechanisms of this association. If processing of social cues is affected by stress exposure, then social cognitive capacities may in part underpin the association between early life stress and psychopathology (10).

Exposure to stress in childhood is associated with aberrant social cognitive abilities, including identification of emotional expressions (10, 11). Studies have reported that exposure to traumatic stress disrupts a child's ability to properly identify or process emotional facial expressions (11, 12). For example, compared to their non-exposed peers, children with a history of maltreatment perform worse at recognizing sad expressions (13, 14). Conversely, another line of research supports the notion that a history of traumatic stress may actually facilitate some types of social-emotional processing. Compared to non-exposed youth, those with traumatic stress exposure are reported to require less perceptual information to identify angry expressions (14, 15), detect fearful expressions at lower intensities (16), and preferentially allocate attention to angry (17) or sad expressions (18). Other studies find no link between stress exposure and emotion processing (19). Moreover, while many studies do not examine sex differences, some report that the effects of stress exposure on social cognition differs in males and females (20, 21). More work is needed to understand if there are sex differences in the effects of stress exposure on social cognition (22).

Studies of healthy youth show that social cognitive capacity improves with age throughout development (23, 24) and is better in females compared to males (25, 26). Traumatic stress exposure and Post Traumatic Stress Disorder (PTSD) have also been linked to social-emotional processing abnormalities (16, 27, 28). Given that stress-related disorders are more prevalent in females (29, 30), research is needed to address the possible roles of age, sex and the presence of PTSD to better understand the links between stress exposure and social

cognition. Such linkage requires examining cognitive and clinical phenotypes in large samples of youth with substantial traumatic stress exposure. The Philadelphia Neurodevelopmental Cohort (PNC) is a unique resource with genetic, clinical, and neurocognitive data from a large community sample of youth, representative of the US urban population (31). We have recently shown that the PNC sample has substantial traumatic stress exposure that is associated with psychopathology (32). This study showed that traumatic stress exposure was associated with lower executive function and complex reasoning efficiency; however, the association with social cognition was more complex. Impairment in social cognitive tasks was evident only at the highest level of trauma exposure, and the linear association between trauma exposure and social cognitive dysfunction did not survive correction for covariates, suggesting a possible sex-specific pattern. Here, we aim to better understand this association and investigate specific social cognitive abilities and sex differences. We hypothesize that high traumatic stress exposure is associated with altered social cognitive performance across the different social cognitive constructs in a sex divergent manner. We evaluated links between high traumatic stress exposure during childhood and adolescence and social cognitive processing abilities in the presence or absence of PTSD.

Methods

Participants

The PNC is a collaboration between the Children's Hospital of Philadelphia and the Brain Behavior Laboratory at the University of Pennsylvania (31). The sample (N=9498) is racially diverse (56% Caucasian, 33% African American and 11% other) and economically diverse (33), and about evenly divided between males and females. Notably, participants were recruited from the pediatric network and not from psychiatric clinics, and the sample is not enriched for individuals seeking psychiatric help.Enrollment criteria included (1) stable health; (2) proficiency in English; (3) physically and cognitively capable of completing interview and neurocognitive assessment; and (4) absence of a disorder that significantly impairs motility or cognition (e.g., paresis or palsy, intellectual disability).After complete description of the study, written informed consent was obtained from participants aged 18, and written assent and parental permission were obtained from children aged<18 and their parents/legal guardian. The University of Pennsylvania and CHOP Institutional Review Boards approved all procedures.

Clinical Assessment including Evaluation of Traumatic Stressful Events (TSE)

Psychopathology symptoms were evaluated using a structured screening interview (GOASSESS), as detailed elsewhere (34), which was based on the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (35). Computerized algorithms used endorsement of symptoms, their frequency and duration, and the presence of distress or impairment to approximate DSM-IV criteria of PTSD, conduct disorder and major depressive episode. Demographics of participants meeting DSM threshold criteria are detailed in supplemental data (Table S1-S3). The GOASSESS TSE screen assessed lifetime exposure to situations in which the participant: (1) experienced a natural disaster or (2) experienced a bad accident; (3) thought that s/he or someone close to him/her was killed or

hurt badly; (4) witnessed someone getting killed, badly beaten, or die; (5) saw a dead body; or was ever her/himself a victim of one of the following assaults: (6) attacked or badly beaten, (7) threatened with a weapon, or (8) sexual assault. For 190 participants (2% of PNC), GOASSESS sections including TSE screening were missing and therefore they were excluded from analyses. In the current study, we compared participants with no TSE exposure (N=5204) to participants with high TSE exposure (endorsement of three or more TSE, N=830, Table 1), which is consistent with prior work (32, 36).

Evaluation of social cognition-related measures

Cognitive assessment used the Penn Computerized Neurocognitive Battery (CNB), a well-established battery, which includes tasks assessing four cognitive domains: executive function, episodic memory, complex reasoning and social cognition (37, 38). The social cognition domain includes three tasks, two assessing central aspects of social-emotional processing and one non-emotional social processing task. The two social emotion processing tasks were Emotion Identification and Emotion Intensity Differentiation. The non-emotion related social task was Age Differentiation.

The Emotion Identification task evaluated participants' ability to accurately identify different emotions. Participants were presented with photographs of faces displaying a happy, sad, angry, fearful, or neutral expression and asked to identify the emotion displayed on each face. Eight pictures of each facial expression were presented, balanced for poser's age, sex, and ethnicity.

The Emotion Intensity Differentiation task measured participants' accuracy in differentiating the intensity of four facial emotional expressions (happy, sad, angry, and fearful). Two expressions of the same poser appeared simultaneously on the screen, both representing the same emotional expression. For each pair, the level of emotional intensity was identical or one face displayed a higher intensity. Participants were asked to indicate which face had the higher level of emotional intensity (e.g., "happier") or indicate that the intensities were identical ("same"). Intensity levels were created by morphing a neutral face with an emotionally intense expression; intensity levels varied from 10–60%. Ten images from each of the four emotional expressions were presented.

The Age Differentiation task was administered to measure participants' ability to discern age differences between two individuals. Two images appeared simultaneously on the screen, both with neutral expressions. Participants were asked to indicate whether the two individuals were the same or different ages, and if different, which was older. The face stimuli were created by morphing young faces into old faces, which resulted in graded levels of age and difficulty. A total of 40 face-pairs were presented. Male and female faces were equally presented.

Statistical Analysis

Given known associations between age and social cognitive performance (39) coupled with the age differences between the high and no TSE exposure groups, linear and non-linear age effects were regressed from all the social cognition outcome measures. As socioeconomic status (SES) is associated with psychopathology (40) and stress exposure also differed

between the two groups, SES was also regressed from all outcome variables. This allowed for the direct assessment of traumatic stress exposure association with social cognitive performance. Of note, the two groups also differed in race, with more minority participants in the high trauma stress exposure group. In the current sample, SES and race were highly collinear. Thus, only one of the two variables (i.e., SES) was used as a covariate in the main set of analyses; however, analyses were repeated regressing out race instead of SES and a similar pattern of findings emerged. Additionally, parallel analyses comparing social cognitive performance between high TSE and no TSE groups matched on sex, age, SES, and race are presented in the supplemental material.

To examine sex differences in the relations between traumatic stress exposure and social emotion processing, we conducted separate two-way analyses of variance (ANOVA) for each task (Emotion Identification, Emotion Intensity Differentiation and Age Differentiation). Group (No TSE exposure, High TSE exposure) and Sex (Male, Female) were between-subject factors, and standardized overall task score was the dependent variable. For each of the three tasks, post-hoc analyses were conducted to compare group performance (High vs. No TSE) in each sex separately. We used a Bonferroni correction (p .025 for all tasks) for multiple comparisons. In the two emotion related tasks that showed sex specific differences between High vs. No TSE Groups, we conducted exploratory analyses comparing accuracy in each emotion (for males in the Emotion Identification task and for females in the Emotion Intensity Differentiation task). We used a Bonferroni correction (p .01 for the Emotion Identification and p .013 for the Emotion Intensity Differentiation) to correct for multiple comparisons of the exploratory analyses. To examine differences in social cognition between high TSE youth with and without PTSD, we performed separate two-way ANOVA for each task (Emotion Identification, Emotion Intensity Differentiation) with Group (No PTSD, PTSD) and Sex (Male, Female) as between subjects' factors, with standardized overall accuracy scores on each task as the dependent variable. Significant Group-by-Sex interactions were probed in planned comparisons that examined group differences on performance for each emotion in males and females. When post-hoc analyses violated the assumption of homogeneity of variance, Welch-Satterthwaite adjustments were used. To better understand the sex-specific findings related to the TSE exposure association with social cognitive performance, analyses were conducted to examine associations between performance in social cognitive tasks with sex specific psychopathology. To do this, we conducted binary logistic regression with psychopathology as the dependent variable (conduct disorder or depressive episode), and the cognitive task performance (accurate identification of angry faces or discrimination between the intensity of happy faces) as the independent variable, controlling for age and SES. A two-tailed p-value less than .05 was considered statistically significant in the omnibus analyses.

Results

Exposure to traumatic stress load

We examined performance on social cognition tasks comparing accuracy between participants with no traumatic stress exposure (No TSE, N=5202) versus high traumatic stress exposure (High TSE, N=830).

Age Differentiation task

Results from the ANOVA for overall accuracy in the Age Differentiation task revealed no main effect of Group, $F(1,5897)=<1$, but there was a main effect of Sex, $F(1,5897)=15.09$, $p<.001$, Cohen's $d=0.23$, such that females (M=.10; SD=.94) outperformed males (M=−.12; SD=1.04). This main effect was qualified by a significant Group-by-Sex interaction, $F(1,5897)=8.28$, $p=.004$. To probe the nature of this interaction, we examined Group differences in performance for males and females. For males, traumatic stress exposure group (M=−.02; SD=1.00) showed a trend to be more accurate at differentiating between ages compared to the control group (M=−.13; SD=1.04), $t(2881)=-2.039$, $p=.04$, Cohen's $d=0.11$. For females, the opposite pattern emerged at trend level, such that the traumatic stress exposure group (M=.02; SD=0.97) tended to be less accurate compared to the control group (M=.12; SD=0.94), $t(3016)=2.03$, $p=.04$, Cohen's $d=0.11$.

Emotion Identification task

Results for overall accuracy on the Emotion Recognition task revealed no significant main effect of Exposure Group, $F(1,5982)=2.27$, $p=.13$, but a main effect of Sex, $F(1,5982)=3.93$, $p=.048$, Cohen's $d=0.13$, where females had higher scores (M=.06; SD=.98) than males (M=−.06; SD=1.02). This effect was qualified by an Exposure Group-by-Sex interaction, $F(1,5982)=4.42$, $p=.036$ (Figure 1). To probe the nature of this interaction, we examined Group differences in performance for males and females. For females, no group difference emerged, $t(3051)=0.43$, $p=.67$. For males, the High TSE group (M=.05; SD=0.89) was more accurate at identifying emotions compared to the No TSE group (M=−.09; SD=1.04), $t(2931)=-2.50$, $p=.013$, Cohen's $d=0.14$.

To better understand which emotions drive the better performance of males in the Exposure Group, we conducted exploratory post-hoc analyses across the five emotions. Males in the High TSE group significantly outperformed the No TSE group on identifying angry expressions, $t(597.43)=2.63$, $p=.009$, Cohen's $d=0.13$, with a trend in the same direction for sad expressions, $t(616.24)=2.50$, $p=.013$, Cohen's $d=0.12$. No other group differences emerged for males, all $p's > .42$. More accurate identification of angry expressions in stress-exposed males was not associated with a bias to inaccurately identify angry expressions when seeing non-angry expressions (see Supplement). Comparison of emotion identification between Exposure Groups matched on sex, age, SES, and race resulted in similar findings (see Supplement).

Emotion Intensity Differentiation task

Results for overall performance on the Emotion Intensity Differentiation task revealed a main effect of Exposure Group, such that the No TSE group (M=.02; SD=1.00) outperformed the High TSE group (M=−.06; SD=1.03), $F(1,5938)=3.91$, $p=.048$, Cohen's $d=0.08$. A main effect of Sex also emerged, $F(1,5938)=12.94$, $p<.001$, Cohen's $d=0.19$, as females (M=.10; SD=.97) outperformed males (M=−.09; SD=1.03). These main effects were qualified by a significant Exposure Group-by-Sex interaction, $F(1,5938)=4.36$, $p=.037$ (see Figure 2). To probe the nature of this interaction, we examined Group differences in performance for males and females. For males, no group difference emerged, $t(2902)=-0.76$, $p=.94$. For females, the High TSE group (M=.03; SD=1.01) was less accurate

discriminating between emotional intensities than the control group (M=.12; SD=0.96), t(3036)= 2.98, p=.003, Cohen's d=0.15.

To better understand which emotions drive the poorer performance of females in the Exposure Group, we conducted exploratory post-hoc analyses across the four emotions. Females in the High TSE group, relative to the No TSE group, were less accurate in differentiating between intensities of happy expressions, t(3036)= 3.31, p=.001, Cohen's d=0.18. There was also a trend for females in the High TSE group to also show reduced accuracy in differentiating between angry expressions, t(3036)= 2.33, p=.02, Cohen's d=0.13, and fearful expressions, t(3036)= 2.14, p=.03, Cohen's d=0.11. No differences were found in females in differentiating sad expressions, p=.18. Comparison of Emotion Intensity Differentiation between the Exposure Groups matched on sex, age, SES, and race resulted in similar findings (see Supplement).

Lifetime PTSD diagnosis in association with performance on social cognition tasks

To assess whether the differences in emotion processing observed in the stress exposed population were attributable to PTSD, we conducted separate analyses only on the participants in the High TSE group, grouping them by a lifetime history of PTSD. We found no differences in the Emotion Identification task or in the Emotion Intensity Differentiation task comparing stress exposed participants with and without a lifetime history of PTSD (PTSD main effect and PTSD-by-Sex interaction, all p's >.05, see supplemental tables S4–5). We found no associations of assaultive traumatic stress exposure (physically, sexually or threatened with a weapon) with altered accuracy social cognitive tasks compared to exposure to non-assaultive stress exposure (main effects for assaultive stress, all p's >.1, all assault by stress interactions p's >.5).

Sex specific association of social cognitive tasks with sex specific psychopathology

To assess whether the sex specific associations we observed between traumatic stress exposure and the social cognitive performance were also associated with sex specific psychopathology, we examined each task's association with psychiatric disorders that have significant sex differences in youth. To do this, we employed binary logistic regressions predicting conduct disorder (more prevalent in males, N= 195, 6.6% of males and N=133, 4.3 % of females) and depressive episode (more prevalent in females, N=379, 12.3% of females and N=235, 7.9% of males).

In males, more accurate identification of angry faces was associated with a diagnosis of conduct disorder (OR 1.175, 95% confidence interval 1.012–1.363, p=.034, corrected for age and SES). No similar association was found for females (OR 1.044, 95% confidence interval 0.878–1.24, p=.562, corrected for age and SES). No association was observed between a diagnosis of a depressive episode and reduced accuracy in discriminating intensity of happy faces in either sex (both p's>.05).

Discussion

In the current study, we report sex-specific alterations in youth with a significant history of traumatic stress exposure in three tasks relevant to social cognition - one non-emotion and

two emotion-processing tasks: (1) Age Differentiation task, in which for males, exposure to multiple traumatic stressors was associated with enhanced performance, whereas females with stress exposure performed worse than non-exposed; (2) the Emotion Identification task, in which stress exposure in males was associated with enhanced ability to recognize angry cues, whereas females with stress exposure did not perform differently than non-exposed; and (3) the Emotion Intensity Differentiation task, where for females, traumatic stress exposure was associated with diminished ability to differentiate between intensities of happy expressions, whereas for males with a history of traumatic stress this ability was spared. Importantly, for those in the high exposure group, these associated changes in emotion processing performance were not associated with a lifetime diagnosis of PTSD, suggesting that high traumatic stress exposure itself is sufficient for the associated emotion processing aberration, even in the absence of a fuller constellation of PTSD symptoms.

The sex differences found in the current study add to the evidence suggesting that there are sex-specific mechanisms associated with stress exposure. For example, prior work has documented sex differences in the effect of stress exposure on brain structure and function (41, 42). Sex differences are also reported in the physiological response to stress (43), with males showing more pronounced cortisol levels and blood pressure changes compared to females when responding to acute stress (43–45). It has been proposed that sex differences in the physiological response to stress may stem from the traditional "fight or flight" stress response having a greater evolutionary advantage for males, priming them to fight or escape threat (44). Speculatively, such sex differences in normative stress responses and threat-associated behaviors may in turn relate to variation in the processing of anger expressions and to the sex-dependent differences in anger identification we see here in stress-exposed youth.

The differential association of stress exposure with social cognition in males and females may also be linked to sex differences in the emergence of psychopathology during and after adolescence. Early life exposure puts females at greater risk for internalizing symptoms and males at risk for externalizing symptoms (46). Thus, underlying social-emotional processing differences in females and males in association with stress exposure might be related to sex specific psychopathology phenotypes. We observed an association between the ability to identify angry faces and diagnosis of conduct disorder in males, and not in females. This finding may suggest that the improved ability of males with high traumatic stress exposure to identify angry faces may be related to the increased prevalence of conduct in males by reducing the threshold for perceived threat, or alternatively that males with conduct symptoms are more likely to incite angry faces in others and therefore "more experienced" in identifying this specific emotion.

By contrast, depression, that is more common in stress-exposed females, has been associated with more difficulties in differentiating between happy facial expressions (47) and the need for higher levels of emotional intensity to recognize happy facial expressions (48). In our study, reduced accuracy in differentiating the intensity of happy faces was found but it was not associated with increased depression risk in females (nor in males). One possible explanation for the differences between the sex specific findings in conduct and depression diagnoses may be due to the clinical nature of these diagnoses and the fact that we assessed

a lifetime history of a depressive episode. While conduct disorder may be viewed as a trait (49), depressive episodes are considered more as states. Therefore, it is possible that participants who endorsed lifetime depressive episodes were not depressed while taking the cognitive tasks, therefore no association was found between the social cognitive performance and the psychopathology measures. Future longitudinal studies are needed to tease apart the temporal relation among stress exposure, social cognitive constructs and various psychopathology phenotypes. In addition, that we found sex by exposure interaction in a non-emotion related social cognitive task raises the possibility that sex specific associations of trauma may be linked more broadly to face processing or social cognition abilities, rather than specifically to emotion processing. This possibility merits further investigation in the context of sex differences in association with trauma exposure.

Traumatic events during brain development are associated with significant cognitive impact (50), most notably described in the field of adult PTSD research in the context of memory and executive function (51–53). The question remains whether the atypical cognitive function is associated with the trauma exposure alone, or with the sequelae of brain changes associated with full blown PTSD diagnosis. A systematic review of studies of adult male veterans suggests that, more significant cognitive deficits are observed in individuals with PTSD compared to individuals who also have traumatic stress exposure but do not have PTSD. This association is less consistent in other clinical populations, with some studies reporting no specific PTSD related cognitive abnormalities beyond the exposure alone (54). There are relatively few studies of the association of trauma with social cognitive capacities. These studies suggest that impaired social cognition may be one mechanism contributing to PTSD risk, for example by reducing the capacity to maintain close social relationships, thus lessening the protective effect of social bonds (55). A recent study conducted in adolescent psychiatric inpatients suggested that social cognition deficits mediate the relationship between insecure attachment and PTSD (56). However, here we did not find differences in social cognitive performance comparing stress-exposed youth with PTSD to those without PTSD. Our results are in line with a study conducted in children reporting an association between maltreatment and atypical processing of emotion that is independent of PTSD diagnosis (16). Taken together, these findings do not support the notion that atypical social cognition in stress-exposed youth is associated with the criterial symptoms of PTSD. Rather, the data suggest that, in non-psychiatric-help-seeking youth, the high traumatic stress exposure itself is associated with specific emotion identification and intensity differentiation patterns - regardless of PTSD. One possible explanation for the inconsistent findings could be that our study population was not clinically ascertained in contrast to the above studies that examined help-seeking populations. Another possibility is an age effect contributing to different findings in adolescents compared to adults with a history of traumatic stress exposure. Finally, the psychopathology screening tool we used may have had reduced sensitivity to detect PTSD compared to studies focused at PTSD that employ more robust tools to assess PTSD diagnosis and severity.

Our study has several limitations. First, the cross sectional nature of the study does not permit causal inference hence we cannot know whether the social cognition abnormalities are due to stress exposure, or if some cognitive traits may put one at increased risk to experience potentially traumatic events, as was suggested previously (57). In addition, we

cannot test whether these abnormalities put youth at risk for the development of PTSD or other stress related phenotypes. Second, the exposure to traumatic events was assessed using a list of eight events, without indication of chronicity or specificity regarding the timing of the traumatic events – each of which may affect the formation of social cognition during childhood and adolescence. The relatively narrow scope of traumatic stressors assessed by the tool limits our ability to directly compare our findings to the available literature relating a broader range of life stressors, including child maltreatment, to social cognitive capacities. Thirdly, although we controlled for such variables as SES and age, other factors (e.g., developmental, demographic, social, or emotional factors) may affect the current results. Future work in this area will need to explore how these other factors may interact with sex and traumatic life events to influence social cognition. Lastly, while our findings show significant sex specific alterations in social cognition related tasks, effect sizes were small. Notwithstanding the small effect sizes, sex specificity of the direction of change associated with high trauma exposure sheds new light on potential brain differences between the sexes that emerges during adolescence. More research is needed to better understand the task- and emotion-specificity of stress-exposure effects on social information processing. The current study only examined social cognition in the context of three tasks; different types of social and emotional processing may yield different patterns of findings.

In summary, this study aimed to elucidate the relationship between traumatic life stress and social cognitive abilities in a large youth cohort including a substantial portion with high traumatic stress exposure. Findings revealed sex-specific associations between exposure and social cognitive capacities. Males exposed to traumatic stress showed a selectively enhanced ability to identify angry facial expressions, which was associated with a diagnosis of conduct disorder, without alterations in identifying other emotions or in differentiating emotional intensity. For females, in contrast, stress exposure was associated with reduced ability to differentiate the intensity of happy emotional expressions, without alterations in differentiating intensity for other emotions or in identifying emotions. That more detrimental and widespread effects of stress exposure did not emerge in the current results is notable and suggests that social cognition may be relatively robust to negative environment influences. This is in contrast to the dose-response negative association between traumatic stress exposure and executive function and complex reasoning that we have previously described in the same cohort (32). Future longitudinal studies examining association between early life trauma and social cognition are needed to delineate the causal pathways.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements:

Abbreviations:

TSE   Traumatic stressful event

PNC   Philadelphia Neurodevelopmental Cohort

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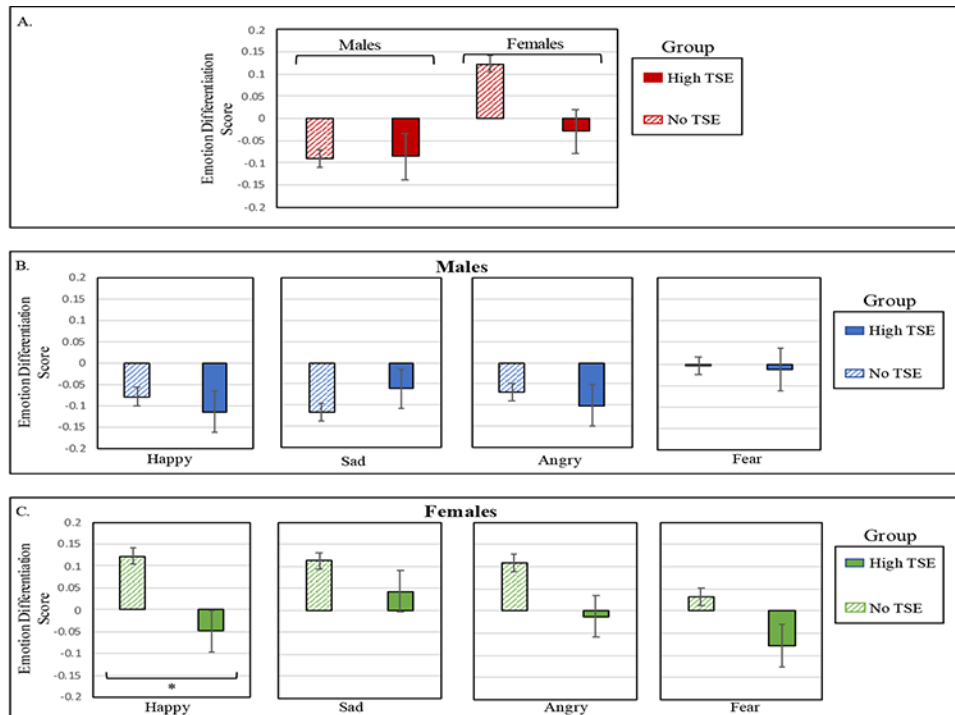
Figure 1. Accuracy performance on Emotion Identification task, by Exposure Group.
Panel A. shows the means and standard errors for performance on Emotion Identification
task accuracy, plotted by Sex and Exposure Group. Performance on each of the separate
emotions are displayed for males (panel B) and females (panel C). In all analyses, age and
race/socioeconomic Z-score were regressed out. TSE=Traumatic Stressful Events. *p .01.
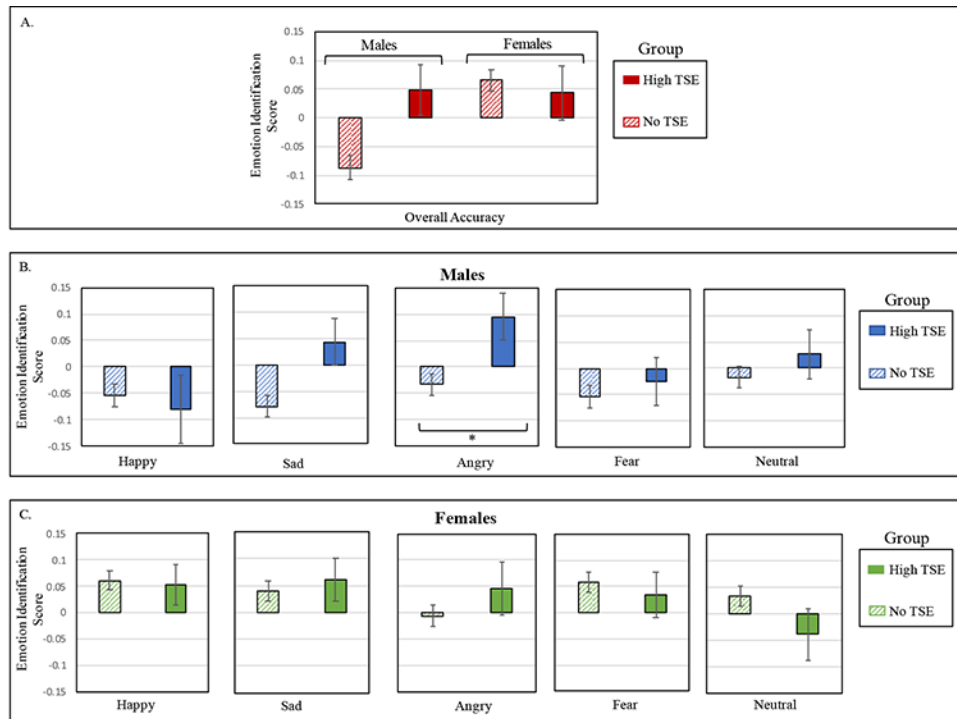
Figure 2. Accuracy performance on Emotion Intensity Differentiation task, by Exposure Group.
Panel A. Means and standard errors for accuracy on the Emotion Intensity Differentiation task, plotted by Sex and Exposure Group. Performance on each of the separate emotions are displayed for males (panel B) and females (panel C). In all analyses, age and race/socioeconomic Z-score were regressed out. TSE=Traumatic Stressful Events. *p .01.

Table 1

Demographic characteristics of study participants.

	No TSE exposure N=5202	High TSE exposure N=830	Test	t/X²	P-value
Age, years (SD)	13.4 (3.6)	16.6 (3)	T-Test	−24.6	<.001
Sex Male, N (%)	2544 (48.9%)	416 (50.1%)	Chi-square	.44	.509
SES z-score (SD)	.16	−.53	T-Test	−19.4	<.001
Caucasian, N (%)	3243 (62.3%)	277 (33.4%)	Chi-square	272	<.001

TSE= Traumatic Stressful Events.