

Archival Report

Sex-Specific Association Between High Traumatic Stress Exposure and Social Cognitive Functioning in Youths

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ABSTRACT

BACKGROUND: Traumatic stressful events (TSEs) during childhood and adolescence are associated with increased risk for psychopathology and cognitive impairment. Aberrations in social cognition may contribute to the psychopathology risk. We examined performance differences on social cognitive measures between youths with high TSE exposure and no TSE exposure and how these effects vary in female and male individuals.

METHODS: The Philadelphia Neurodevelopmental Cohort investigates clinical and cognitive phenotypes in a U.S. youth (aged 8–21 years) community population. Here we compared performance in social cognition tasks between youths with high exposure (≥ 3 TSEs, $n = 830$) and youths 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, male subjects with high traumatic stress exposure outperformed nonexposed male subjects; exposure did not affect performance in female subjects. In the emotion intensity differentiation task, female subjects with high traumatic stress exposure performed worse than nonexposed female subjects, with no difference in male subjects between exposure groups. Exploratory analyses revealed that sex differences were driven by improved identification of angry expressions in stress-exposed male subjects and poorer performance in differentiating intensity of happy expressions in stress-exposed female subjects.

CONCLUSIONS: 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: Childhood trauma, Developmental psychopathology, Emotion processing, Sex differences, Social cognition, Traumatic stress

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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 during 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 with their nonexposed 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 with nonexposed youths, 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 (18) expressions. 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 differ in male and female individuals (20,21). More work is needed to understand whether there are sex differences in the effects of stress exposure on social cognition (22).

Studies of healthy youths show that social cognitive capacity improves with age throughout development (23,24) and is better in female youths compared with male youths (25,26). Traumatic stress exposure and posttraumatic stress disorder (PTSD) have also been linked to social-emotional processing abnormalities (16,27,28). Given that stress-related disorders are more prevalent in female individuals (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 youths 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 youths representative of the U.S. urban population (31). We recently showed that the PNC sample has substantial traumatic stress exposure that is associated with psychopathology (32). The 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 aimed to better understand this association and investigate specific social cognitive abilities and sex differences. We hypothesized 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 AND MATERIALS

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 it is about evenly divided between male and female individuals. 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 were 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 years and written assent and parental permission were obtained from children aged < 18 and their parents/legal guardians. The University of Pennsylvania and Children's Hospital of Philadelphia Institutional Review Boards approved all procedures.

Clinical Assessment Including Evaluation of Traumatic Stressful Events

Psychopathology symptoms were evaluated using a structured screening interview (GOASSESS, based on the Grand Opportunity National Institute of Mental Health mechanism of funding), as detailed elsewhere (34), which was based on the Kiddie Schedule for Affective Disorders and Schizophrenia (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 Tables S1–S3. The GOASSESS traumatic stressful event (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 he/she or someone close to him/her was going to get killed or hurt badly; 4) witnessed someone getting killed, getting badly beaten, or dying; 5) saw a dead body or was ever himself/herself a victim of one of the following assaults: 6) attacked or badly beaten, 7) threatened with a weapon, or 8) sexually assaulted. 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 ($n = 5204$) with participants with high TSE exposure (endorsement of three or more TSEs; $n = 830$; Table 1), which is consistent with prior work (32,36).

Evaluation of Social Cognition-Related Measures

Cognitive assessment used the Penn Computerized Neurocognitive Battery, a well-established battery that 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 tasks assessing central aspects of social-emotional processing and one nonemotional social processing task. The two social emotion processing tasks were emotion identification

Table 1. Demographic Characteristics of Study Participants

	No TSE Exposure ($n = 5202$)	High TSE Exposure ($n = 830$)	t/χ^2	p Value
Age, Years (SD)	13.4 (3.6)	16.6 (3)	$t = -24.6$	$< .001$
Sex, Male, n (%)	2544 (48.9)	416 (50.1)	$\chi^2 = 0.44$.509
SES Z Score	0.16	-0.53	$t = -19.4$	$< .001$
Race, Caucasian, n (%)	3243 (62.3)	277 (33.4)	$\chi^2 = 272$	$< .001$

SES, socioeconomic status; TSE, traumatic stressful event.

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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 were asked to identify the emotion displayed on each face. Eight pictures of each facial expression were presented, balanced for posers' 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 to 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% to 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 nonlinear age effects were regressed from all the social cognition outcome measures. Because 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. In addition, parallel analyses comparing social cognitive performance between high-TSE and no-TSE groups matched on sex, age, SES, and race are presented in the [Supplement](#).

To examine sex differences in the relations between traumatic stress exposure and social emotion processing, we conducted separate two-way analyses of variance for each task (emotion identification, emotion intensity differentiation, and age differentiation). Group (no TSE exposure or high TSE exposure) and sex (male or 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 TSE vs. no TSE) in each sex separately. We used a Bonferroni correction ($p \leq .025$ for all tasks) for multiple comparisons. In the two emotion-related tasks that showed sex-specific differences between high-TSE and no-TSE groups, we conducted exploratory analyses comparing accuracy in each emotion (for male subjects in the emotion identification task and for female subjects in the emotion intensity differentiation task). We used a Bonferroni correction ($p \leq .01$ for the emotion identification task and $p \leq .013$ for the emotion intensity differentiation task) to correct for multiple comparisons of the exploratory analyses. To examine differences in social cognition between high-TSE youths with and without PTSD, we performed separate two-way analyses of variance for each task (emotion identification or emotion intensity differentiation) with group (no PTSD or PTSD) and sex (male or 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 male and female subjects. 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; $n = 5202$) and those with high traumatic stress exposure (high TSE; $n = 830$).

Age Differentiation Task. Results from the analysis of variance 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 female subjects (mean = 0.10, SD = 0.94) outperformed male subjects (mean = -0.12, 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 male and female subjects. For male subjects, the traumatic stress exposure group (mean = -0.02, SD = 1.00) showed a trend to be more accurate at differentiating between ages compared with the control group (mean = -0.13,

SD = 1.04), $t_{2881} = -2.039$, $p = .04$, Cohen's $d = 0.11$. For female subjects, the opposite pattern emerged at trend level, such that the traumatic stress exposure group (mean = 0.02, SD = 0.97) tended to be less accurate compared with the control group (mean = 0.12, 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 female subjects had higher scores (mean = 0.06, SD = 0.98) than male subjects (mean = -0.06, 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 male and female subjects. For female subjects, no group difference emerged, $t_{3051} = 0.43$, $p = .67$. For male subjects, the high-TSE group (mean = 0.05, SD = 0.89) was more accurate at identifying emotions compared with the no-TSE group (mean = -0.09, SD = 1.04), $t_{2931} = -2.50$, $p = .013$, Cohen's $d = 0.14$.

To better understand which emotions drive the better performance of male subjects in the exposure group, we conducted exploratory post hoc analyses across the five emotions. Male subjects in the high-TSE group significantly outperformed those in 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 male subjects, all $ps > .42$. More accurate identification of angry expressions in stress-exposed male subjects was not associated with a bias to inaccurately identify angry expressions when seeing nonangry 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 (mean = 0.02, SD = 1.00) outperformed the high-TSE group (mean = -0.06, 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 female subjects (mean = 0.10, SD = 0.97) outperformed male subjects (mean = -0.09, 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 male and female subjects. For male subjects, no group difference emerged, $t_{2902} = -0.76$, $p = .94$. For female subjects, the high-TSE group (mean = 0.03, SD = 1.01) was less accurate in discriminating between emotional intensities than the control group (mean = 0.12, SD = 0.96), $t_{3036} = 2.98$, $p = .003$, Cohen's $d = 0.15$.

To better understand which emotions drive the poorer performance of female subjects in the exposure group, we conducted exploratory post hoc analyses across the four emotions. Female subjects in the high-TSE group, relative to those in 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 female subjects 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 female subjects 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).

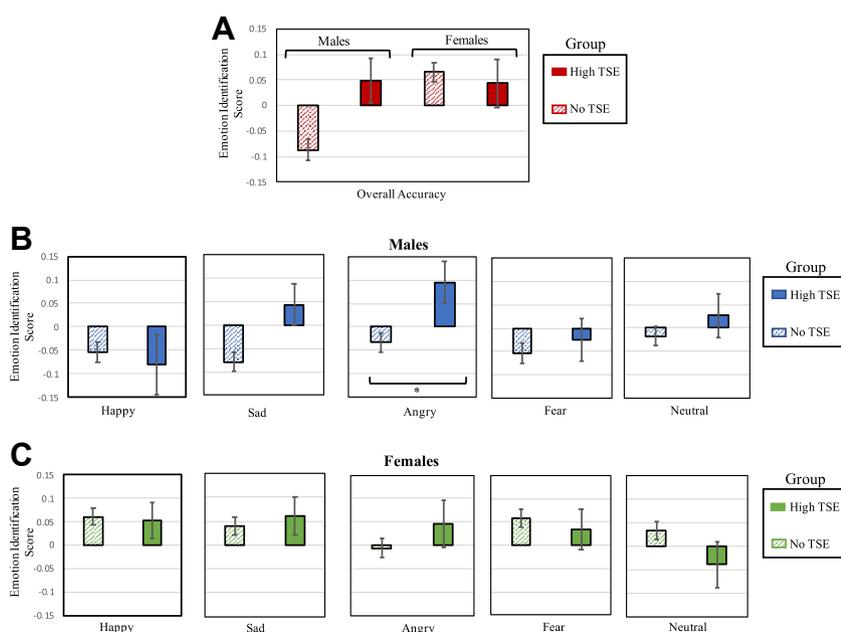


Figure 1. Accuracy performance on emotion identification task by exposure group. (A) Means and standard errors for performance on emotion identification task accuracy plotted by sex and exposure group. Performance on each of the separate emotions is displayed for male subjects (B) and female subjects (C). In all analyses, age and race/socioeconomic Z score were regressed out. * $p \leq .01$. TSE, traumatic stressful events.

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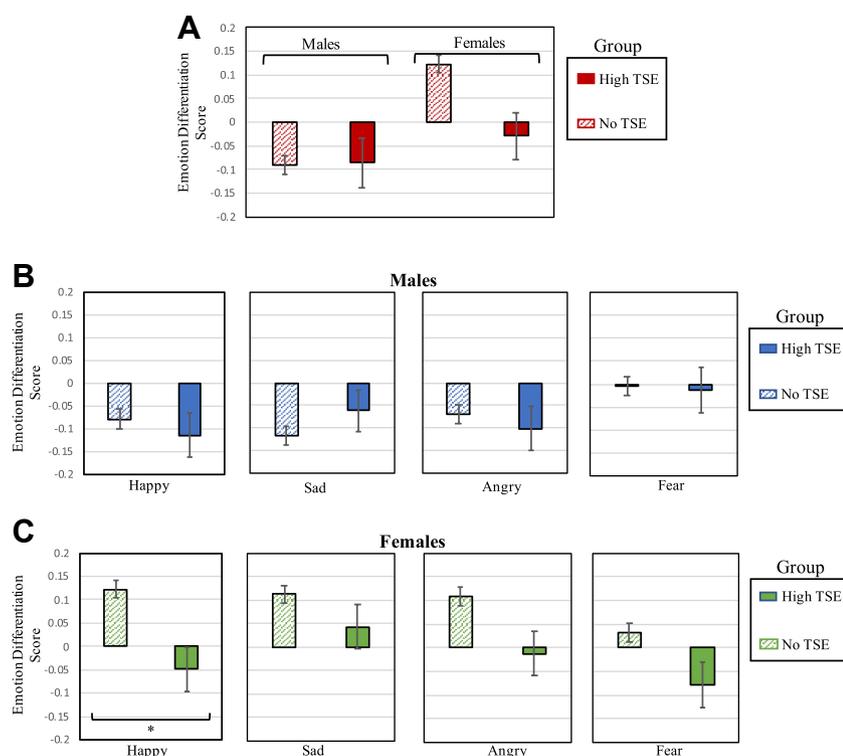


Figure 2. Accuracy performance on emotion intensity differentiation task by exposure group. **(A)** Means and standard errors for accuracy on the emotion intensity differentiation task plotted by sex and exposure group. Performance on each of the separate emotions is displayed for male subjects **(B)** and female subjects **(C)**. In all analyses, age and race/socioeconomic Z score were regressed out. * $p \leq .01$. TSE, traumatic stressful events.

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 and S5). We found no associations of assaultive traumatic stress exposure (physically, sexually, or threatened with a weapon) with altered accuracy in social cognitive tasks compared with exposure to non-assaultive stress exposure (main effects for assaultive stress, all p s $> .10$, all assault by stress interactions, p s $> .50$).

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 youths. To do this, we employed binary logistic regressions predicting conduct disorder (more prevalent in male subjects; $n = 195$, 6.6% of male subjects and $n = 133$, 4.3% of female subjects) and depressive episode (more prevalent in female subjects; $n = 379$, 12.3% of female subjects and $n = 235$, 7.9% of male subjects).

In male subjects, more accurate identification of angry faces was associated with a diagnosis of conduct disorder (odds ratio = 1.175, 95% confidence interval = 1.012–1.363, $p = .034$, corrected for age and SES). No similar association was found for female subjects (odds ratio = 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 youths with a significant history of traumatic stress exposure in three tasks relevant to social cognition, one nonemotion task and two emotion processing tasks: 1) the age differentiation task, in which exposure to multiple traumatic stressors in male subjects was associated with enhanced performance, whereas female subjects with stress exposure performed worse than nonexposed ones; 2) the emotion identification task, in which stress exposure in male subjects was associated with enhanced ability to recognize angry cues, whereas female subjects with stress exposure did not perform differently than nonexposed ones; and 3) the emotion intensity differentiation task, in which traumatic stress exposure in female subjects was associated with diminished ability to differentiate between intensities of happy expressions, whereas for male subjects 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 male subjects showing more pronounced cortisol levels and blood pressure changes compared with female subjects 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 male individuals, 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 youths.

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

By contrast, depression, which is more common in stress-exposed female individuals, 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 female subjects (or in male subjects). One possible explanation for the differences between the sex-specific findings in conduct and depression diagnoses may be 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 performing the cognitive tasks and, 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 a 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 with 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, such as by reducing the capacity to maintain close social relationships, thereby 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 youths with PTSD with 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 youths is associated with the criterial symptoms of PTSD. Rather, the data suggest that, in nonpsychiatric-help-seeking youths, 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 with adults with a history of traumatic stress exposure. Finally, the psychopathology screening tool we used may have had reduced sensitivity to detect PTSD compared with studies focusing on 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 whether some cognitive traits may put one at increased risk for experiencing potentially traumatic events, as was suggested previously (57). In addition, we cannot test whether these abnormalities put youths 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

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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 with the available literature relating a broader range of life stressors, including child maltreatment, to social cognitive capacities. Third, although we controlled for such variables as SES and age, other factors (e.g., developmental, demographic, social, and 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. Last, 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 examined social cognition only 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. Male subjects 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 female subjects, 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 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.

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RB serves on the scientific board and reports stock ownership in Taliaz Health with no conflict of interest relevant to this work. All other authors report no biomedical financial interests or potential conflicts of interest.

ARTICLE INFORMATION

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